A STUDY ON NEW ONSET ARRHYTHMIAS IN ACUTE MYOCARDIAL INFARCTION

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&

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BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"A STUDY ON NEW ONSET ARRHYTHMIAS IN ACUTE MYOCARDIAL INFARCTION"** is a bonafide original work of **DR.V.R.BHUVANESWARI** in partial fulfilment of the requirements of M.D General Medicine [Branch-1] examination of The Tamilnadu Dr.M.G.R Medical University to be held in MAY - 2018.

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DECLARATION

I solemnly declare that the dissertation titled **"A STUDY ON NEW ONSET ARRHYTHMIAS IN ACUTE MYOCARDIAL INFARCTION"** is done by me at K.A.P.VISWANATHAM GOVTMEDICAL COLLEGE, TIRUCHIRAPALLI-1 under the guidance and supervision of PROF.DR.V.USHA PADMINI, M.D. This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University towards the partial fulfilment of the requirements for the award of M.D Degree [Branch-1] in General Medicine.

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CONTENT

INTRODUCTION

Acute myocardial infarction is one of the commonest emergencies in the developed and developing countries. Death from arrhythmias especially ventricular tachycardia has been one of the commonest causes of sudden cardiac death following acute myocardial infarction. In the prefibrinolytic era, deaths reported after acute MI were as high as 60 percent, which is usually seen within first twenty four hours, especially in the first hour. This high death rate was attributable to usually ventricular fibrillation[1].

However in recent years, improvements in the diagnosis and treatment modalities has improved the outcomes associated with acute MI including the outcome of ventricular arrhythmias that occur following acute MI. These has led to significant fall in mortality associated with the complications of acute MI.

Many studies have been done to evaluate the incidence of arrhythmias complicating the peri-infarct period, to study their prognostic significance and outcomes with various treatment modalities[2]. But comparison of these studies is not easy because of the different study populations, different types of infarct, and also the variations in the types of arrhythmias reported. Also there is not much documented evidence regarding these periventricular arrhythmias in the population of Trichy, Tamilnadu. Thus the purpose of this study is to study the incidence, the profile of arrhythmias and the outcome associated with them in the population of Trichy.

AIMS AND OBJECTIVES

1. To study incidence and profile of various types of new onset arrhythmias in acute MI- both STEMI and NSTEMI
2. To study various types of arrhythmias in relation to the wall involved
3. To study various types of arrhythmias in relation to time between the admission and the onset.

MATERIALS AND METHODS

SOURCE OF DATA

The present study was done at Mahatma Gandhi Memorial Govt. Hospital attached to K.A.P.V.Govt.Medical College, Tiruchirapalli.

STUDY DESIGN

Cross-sectional prospective study.

PERIOD OF STUDY

The study was conducted from June 2016 to June 2017.

INCLUSION CRITERIA

1) Patients with ECG changes suggestive of acute MI

2) Patients presenting with in 24 hours of onset of symptoms suggestive of acute MI

3) Acute MI patients lysed or not presenting within 24 hours of onset of illness

4) Willingness of the patient

EXCLUSION CRITERIA

1) Patients with past history of MI

2) Patients with coronary artery disease on drugs

3) Patients who are known cases of arrhythmias on treatment.

4) Patients with structural defects in the heart like Rheumatic Heart diseases and Congenital Heart diseases

5) Patients without willingness to participate

ETHICS COMMITTEE APPROVAL

Approval was obtained from Institutional Ethics Committee.

CONSENT

Informed consent was obtained from all the participants and their relatives wherever necessary.

PROCEDURE OF STUDY

In this study,100 cases of acute myocardial infarction presenting to the emergency room of Mahatma Gandhi Memorial Government Hospital, Trichy attached to KAPV Government Medical College were studied for occurrence of various new onset arrhythmias in relation to typ e of MI,wall involved and time duration of illness.

Patients were included according to the criterias mentioned above, after getting informed consent. These patients were closely monitored with serial ECG done at 1,3,6,12,24,48,72 hours. Various investigations like urea, sugar,creatinine, serum cholesterol, serum electrolytes, cardiac enzymes were done and assessed.

ECG parameters like heart rate, rhythm, p wave morphology, qrs morphology, t wave morphology, PR interval, ST segment, QT interval, p wave axis, qrs axis, chamber hypertrophy / dilatation, were assessed periodically and noted.

STATISTICAL ANALYSIS

All the parameters were tabulated. Mean, Standard deviation were analysed using SPSS 20 software. All the biochemical parameters were correlated with serum cholinesterase using intercorrelations. Chi-square test was the test of significance used

for qualitative variables to find the association between them. T test was the test of significance used for comparing quantitative variables with qualitative variable. One-way Anova is used as test of significance to assess various parameters with the compound used for poisoning.

REVIEW OF LITERATURE

Complications of MI may be classified as mechanical, arrhythmic, inflammatory (early pericarditis and post-MI syndrome) sequelae, and left ventricular mural thrombus (LVMT). Other fatal complications are right ventricular (RV) infarction and cardiogenic shock[3].

ARRHYTHMIAS COMPLICATING MI

Arrhythmias following MI is more common immediately after or during the event. Serious arrhythmias like ventricular fibrillation occurs more in the first hour following the event, later the incidence decreases.ST elevation MI has more risk of arrhythmias than Non ST Elevation myocardial infarction.

Usually peri- infarct arrhythmias are benign and self limiting. At times it can cause hypotension,increase myocardial oxygen requirements and can lead to fatal ventricular arrhythmias. These should be monitored and treated aggressively[4].

PATHOPHYSIOLOGY OF ARRHYTHMIAS OCCURRING IN ACUTE MYOCARDIAL INFARCTION

Generalized autonomic dysfunction the myocardium in acute myocardial infarction leads to enhanced automaticity of the myocardium and conduction system which leads to arrhythmias. Electrolyte imbalances and hypoxia also contribute to arrhythmias. The damaged myocardium is more prone for re-entrant circuit, making it more susceptible for arrhythmias.

Enhanced efferent sympathetic activity, increased catecholamines, play a role in the pathogenesis of peri-infarction arrhythmias.Transmural infarction interrupt sympathetic flow to the myocardium distal to the area of infarction[5].

CLASSIFICATION OF PERI-INFARCTION ARRHYTHMIAS

Peri-infarction arrhythmias can be broadly classified into the following categories:

- Bradyarrhythmias, including sinus bradycardia and junctional bradycardia
- Atrioventricular (AV) blocks, including first-degree AV block, second-degree AV block, and third-degree AV block
- Intraventricular blocks, including left anterior fascicular block, right bundle branch block (RBBB), and left bundle branch block (LBBB)
- Supraventricular tachyarrhythmias, including sinus tachycardia, premature atrial contractions, paroxysmal supraventricular tachycardia, atrial flutter, and atrial fibrillation
- Accelerated junctional rhythms
- Ventricular arrhythmias, including premature ventricular contractions (PVCs), accelerated idioventricular rhythm, ventricular tachycardia, and ventricular fibrillation
- Reperfusion arrhythmias[6].

SUPRAVENTRICULAR TACHYARRHYTHMIAS

Increased sympathetic activity plays an important role in development of sinus tachycardia which can result in hypertension or hypotension. This leads to increased

myocardial oxygen demand and worsens myocardial ischaemia by decreasing the diastole. Persistent tachycardia may be due to pain, anxiety, anemia, heart failure, hypoxia, hypovolemia, pulmonary embolism, pericarditis.

Sinus tachycardia following acute MI should be identified earlier and treated appropriately because it worsens the ischaemia. Treatment includes use of beta – blockers or nitrates to relieve ischaemia, adequate pain management, oxygenation, volume repletion for hypovolemia, administration of anti- inflammatory agents to treat pericarditis, dieresis to manage heart failure[7].

PREMATURE ATRIAL CONTRACTIONS

Premature atrial contractions often precede paroxysmal supraventricular tachycardia, atrial flutter, or atrial fibrillation . These extra impulses are usually due to atrial distention causing increased left ventricular (LV) diastolic pressure or inflammation associated with pericarditis. No specific therapy is indicated[8].

PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA

Incidence of PSVT in the setting of AMI is less than 10 %. In hemodynamically stable patients , adenosine can be used. In patients without LV failure, i.v. diltiazem or a beta – blocker can be used. In hemodynamically unstable patients , synchronized electrical cardioversion is required[9].

ATRIAL FLUTTER

Incidence of atrial flutter is only less than 5 %. Treatment strategy is similar to atrial fibrillation. Synchronised electrical cardioversion may be needed in case of decreased coronary blood flow and or hemodynamic compromise. In case of refractory atrial flutter, overdrive atrial pacing is considered[10].

ATRIAL FIBRILLATION

Incidence of atrial fibrillation is 10 – 15 %. Causes include LV failure, RV infarction, pericarditis ,ischaemic injury to the atria, conditions causing elevated left atrial pressure. Risk of mortality and stroke increases in AF following acute MI. If the patient is unstable immediate electrical cardioversion is indicated. Synchronized electrical cardioversion with 200 J is used.Continuous sedation or general anesthesia should be given before the procedure.

If the patient is not responding to cardioversion i.v. amiodarone or i.v. digoxin can be used. In case of stable patients, rate control is achieved by using i.v.metoprolol or i.v. diltiazem. Diltiazem should not be used in patients with moderate – severe heart failure. Conversion to sinus rhythm is considered in patients with new onset sustained tachycardia. Anticoagulation with unfractionated heparin or LMWH is considered in patients with atrial flutter and fibrillation, to reduce the risk of thromboembolism[11].

ARRHYTHMIC COMPLICATIONS: ACCELERATED JUNCTIONAL RHYTHM

Accelerated junctional rhythm Uaually seen following inferior wall MI. It is a result of increased automaticity of the junctional tissue, with a heart rate of 70- 130 bpm. Treatment is directed at correcting the underlying ischaemia.

ARRHYTHMIC COMPLICATIONS: BRADYARRHYTHMIAS

SINUS BRADYCARDIA

Inferior or posterior acute myocardial infarction is commonly associated with sinus bradycardia. Highest incidence is seen in the first two hours following the event. Stimulation of cardiac vagal afferent receptors results in efferent cholinergic stimulation of the heart which leads to sinus bradycardia. If untreated may lead to reduced cardiac output and hypotension , which may result in ventricular arrhythmias. It is not associated with increase in risk of mortality. Therefore therapy is indicated only when adverse signs or symptoms is present.

In case of emergency, (HR <40 bpm, hypotension,) i.v. atropine sulphate is given at a dose of 0.5 – 1mg every 3 – 5 minutes to a maximum of 0.03–0.04mg/kg . If the hypotension does not reverse volume depletion or RV infarction is considered. If atropine therapy is ineffective, transcutaneous or transvenous pacing is indicated. Denervated , transplanted hearts are non responsive to atropine, therefore cardiac pacing is needed. Dopamine 5-20 mcg/kg/min given intravenously, epinephrine 2-10 mcg/min, and/or dobutamine may be used as additional therapy[12].

JUNCTIONAL BRADYCARDIA

Junctional bradycardia is a protective AV junctional escape rhythm at a rate of 35-60 bpm in patients who have an inferior MI. This arrhythmia is not usually associated with hemodynamic compromise, and treatment is typically not required.

ARRHYTHMIC COMPLICATIONS: AV AND INTRAVENTRICULAR BLOCKS

FIRST-DEGREE AV BLOCK

First degree AV block is most common in acute inferior wall myocardial infarction. Incidence of first degree AV block in AMI is 15 %. First degree AV block is defined as prolongation of PR interval more than 0.20 seconds. Usually patients with first degree AV block have conduction disturbances above the bundle of His. Treatment is indicated only when associated with hemodynamic compromise. Progression of first degree AV block to ventricular asystole or complete heart block is rare.

In case of Calcium channel blockers and beta-blocker usage, stopping the drug is considered if hemodynamic impairment or progression of the block occurs. Atropine is administered and continuous cardiac monitoring is done[13].

SECOND-DEGREE AV BLOCK

Incidence of second degree AV block (Mobitz type I or II) following Acute MI is 10% . Second degree AV block is usually associated with inferior wall MI . Mobitz typeI AV block accounts for 90% of all second degree AV block following

MI. Treatment is not always needed for Mobitz type I AV block. If the heart rate is very low, 0.5-1 mg atropine is administered intra venously . Rarely transcutaneous or transvenous pacing is required.

Mobitz type II AV block accounts for 10% of all second degree AV block . Mobitz type II block is usually associated with anterior wall MI. Second degree AV block often progresses to complete heart block. Mor tality is high in Mobitz II AV block , as it can progress to Complete heart block. Therefore immediate treatment is needed with transcutaneous pacing or atropine. A temporary transvenous pacemaker or a permanent pacemaker will be the final solution[14].

THIRD-DEGREE AV BLOCK

Incidence of third degree AV block following acute MI is 5 – 15%. Complete heart block develop gradually from first or second degree heart blocks in patients with anterior or inferior infarctions.If the block is supranodal or intranodal , the escape rhythm usually has narrow QRS,with rates exceeding 40 bpm. If the block is below the His bundle,the escape rhythm has wide QRS and the rate is slower than 40 bpm.

Complete heart block occurring in inferior wall MI is responsive to atropine. These patients may recover without need for a pacemaker. Complete heart block following isolated inferior wall MI has a mortality of 15%. In case of coexisting RV MI mortality is higher. Immediate treatment with atropine is indicated. If the patient is unresponsive, temporary transcutaneous or transvenous pacing is indicated. Permanent pacing may be needed in patients who do not respond to thrombolysis or PCI.

Complete heart block occurring in acute anterior wall MI, is usually preceeded by an intraventricular block or a Mobitz type II block. Immediate treatment with atropine or transcutaneous pacing may be needed. Transvenous pacing may be required in some patients. Permanenent pacemaker is the ultimate treatment in patients with complete heart block following anterior wall MI[15].

INTRAVENTRICULAR BLOCKS

His bundle divides into 3 fascicles and supply the ventricles, the anterior division of the left bundle, the posterior division of the left bundle, and the right bundle. Intraventricular blocks occurring in one of these fascicles constitute for 15% of the conduction disturbances occurring in acute MI. Incidence of LAFB is 3-5 %, LAFB is only 1-2 %. Posterior fascicle block usually occurs in large MI and has high mortality rate.

Incidence of RBBB following acute MI is 2%. Since the right bundle receives blood supply from the Left anterior descending artery, a new RBBB suggests a large infarct territory. It is a major risk factor for cardiogenic shock, due to large size of the infarct. Proximal LAD occlusion is charecterised by bifascicular block, combination of RBBB and LAFB. Bifascicular block does not usually progress to complete heart block. When it occurs along with first degree AV block, it is called a trifascicular block. Progression of trifascicular block to complete heart block occurs in 40% of the patients[16].

ARRHYTHMIC COMPLICATIONS: VENTRICULAR ARRHYTHMIAS

PREMATURE VENTRICULAR CONTRACTIONS

Premature ventricular contractions is commonly observed in post MI patients. But these arrhythmias usually do not develop into ventricular fibrillation.primary ventricular fibrillation arises denovo. Prophylactic suppression of PVCs is not recommended. On the contrary prophylactic suppression has increased risk of asystole or bradycardia. Therefore treatment is not aimed at giving antiarrhythmics, but on correcting any associated electrolyte or metabolic abnormalities[17].

ACCELERATED IDIOVENTRICULAR RHYTHM

Incidence of accelerated idioventricular rhythm is 20 % following acute MI . Accelerated idio ventricular rhythm is characterized by wide QRS ventricular rhythm with the regular escape rate faster than atrial rate but less than 100 beats per minute. Slow non conducted P waves unrelated to the QRS complexes may be seen indicating AV dissociation .

Usually the episodes are short and spontaneously terminating . Incidence is equal in anterior and inferior infarctions . The pathogenesis may be a structural damage to the SA node or the AV node or a ventricular ectopic focus that takes over as a dominant pace maker .

AIVR does not have a prognostic value . Untreated cases do not show increase in the incidence of VF or mortality. This rhythm is more frequently seen in patients who develop early reperfusion. But this rhythm cannot be used as a marker of reperfusion. In symptomatic patients who develop hypotension or ischaemia temporary pacing may be indicated. Suppression of this rhythm can result in

significant bradycardia or asystole. Therefore an accelerated idioventricular rhythm should be left untreated[18].

NONSUSTAINED VENTRICULAR TACHYCARDIA

It is defined as three or more ventricular ectopic beats occurring at a rate greater than 100 bpm and lasting for less than 30 seconds. In case of repeated episodes, sudden hemodynamic collapse can occur. Nonsustained VT occurring within 48 hours of acute MI does not increase the mortality.Antiarrhythmic treatment does not offer a benefit. However nonsustained VT occurring after 48 hours of acute MI has increased of sudden cardiac death. Treatment is mandatory in these patients. Electrolyte disturbances should be treated promptly[19].

SUSTAINED VENTRICULAR TACHYCARDIA

Sustained VT is characterized by 3 or more consecutive ventricular ectopics occurring at a rate greater than 100 bpm and lasting longer than 30 seconds. Myocardial scar gives rise to monomorphic VT. Polymorphic VT responds to treatment of cardiac ischemia. Mortality rate is as high as 20 % in sustained polymorphic VT. Sustained VT is an emergency, since it can easily deteriorate into ventricular fibrillation.

In patients with hemodynamically unstable polymorphic VT, immediate unsynchronized DC cardioversion of 200 J should be given. However monomorphic VT should be treated with synchronized DC cardioversion of 100 J. If the patient is hemodynamically stable, antiarrhythmic therapy is considered before cardioversion. Associated electrolyte disturbances, acid- base disturbances ,hypoxia should be

corrected. In patients with persistent or recurrent VT overdrive pacing can restore sinus rhythm[20].

VENTRICULAR FIBRILLATION

Incidence of primary VF is 4.5% in the first hour, 60% within 4 hours, 80 % within 12 hours following MI. Secondary VF associated with cardiogenic shock has very high mortality rate of 40 – 60 %. Usually secondary VF occurs more than 48 hours following MI. Complications like pump failure and cardiogenic shock sre common.

VF needs emergency treatment with unsynchonized DC shock of 200 – 300 J as early as possible. Extensive myocardial ischemia, necrosis or cardiac rupture should be considered if there is no effective contraction evenafter restoration of synchronous cardiac electrical activity. Antiarrhythmics like amiodarone or lidocaine helps to prevent recurrent or refractory episodes. Continuous intravenous infusion of antiarrhythmics for 12 – 24 hrs after electrical cardioversion improves the success rate of treatment.

There is no role of prophylactic lidocaine infusion, because of associated complications like bradycardia and asystole. Early use of beta – blockers is recommended in acute MI to reduce the risk of VF [21].

ARRHYTHMIC COMPLICATIONS: REPERFUSION ARRHYTHMIAS

It was believed that new onset arrhythmias following acute MI are a marker of successful coronary reperfusion. But these arrhythmias are also seen in patients in

whom reperfusion is unsuccessful. Therefore these reperfusion arrhythmias cannot be used as a marker of coronary reperfusion and it should be treated like AIVR.

Arrhythmias are well recognized complication of acute myocardial infarction. Acute MI is characterized by enhanced automaticity in ischemic zone, increased tissue excitability, regional dispersion of repolarization. These changes play an important role in the pathogenesis of ventricular arrhythmias. Incidence of ventricular arrhythmias following acute MI is 2 – 20 %.

Initially it was believed that ventricular arrhythmias following acute MI does not affect the outcome. Subsequent RCTs showed that sustained ventricular arrhythmias may be associated with unresolved occlusion of the artery and may be associated with early mortality[22].

Sustained ventricular arrhythmias are associated with increased mortality inspite of thrombolytic therapy. It remains a controversy whether patients undergoing mechanical revascularization ,who develop arrhythmias are at increased risk of adverse outcome.

In a study by Jasim et al in 2003 at Ibna – Sina Teaching Hospital in mosul,Iraq, 618 patients admitted to CCU were studied. MI with heart block was seen in 61 (9.9%) patients, of them 14 died (22.9%). In the control group was 60 patients with acute MI without heart block, 5 of them died (8.3%). These evidence show that prognosis is affected by the presence of heart block. And the mortality increases with progressing degree of heart block, with maximum risk for complete heart block[23].

In a study by Verma Y et al in 2001, 310 patients admitted to ICCU in Gandhi medical college,Bhopal, were studied for the territory wise incidence of acute

myocardial infarction, conduction disturbances associated and the effect of thrombolysis on prognosis. As per accepted ECG criterias,Territory-wise incidence of acute MI was per anteroseptal MI 37%, anterior wall MI 20%, extensive anterior wall MI 15%, inferior wall MI 28%. Incidence of conduction disturbance was 20% with a distribution of: right bundle branch block (RBBB) - 58%, complete heart block (CHB)-21%, left bundle branch block (LBBB)-12%, left anterior fascicular block (LAFB)- 5.5% and Wenkebach's - 3.5%. 25 patients of AMI with fresh bifascicular block or RBBB were studied .The overall mortality in AMI with RBBB was 40%. 23 % mortality was seen in thrombolyzed patients with RBBB and 60 % mortality was seen in non thrombolyzed patients with RBBB. It was therefore concluded that significantly high mortality was seen among patients of AMI presenting with RBBB and mortality is even higher in the sub group of nonthrombolyzed patients[24].

In a study by Archbold RA et al in 1998, which was conducted in CCU of new Ham General Hospital UK, 1225 patients were studied. Incidence of conduction defects was 16%. 65 cases of AV nodal block (5.3%) , 29 cases of LBBB (2.4%) and 44 cases of RBBB (3.6%) and 36 cases of bifascicular block(2.9%) and 20 cases of complete heart block (CHB) (1.6%) was recorded. Percentage of mortality in patients with conduction defects was as follows : 19% in patients with normal conduction, 38% in patients with RBBB, 57% in patients with LBBB, 58% in patients with bifascicular block and 60% in patients with complete heart block[25].

In a study by David T G et al in 1988, 164 patients of acute MI admitted to CCU of medical college Ludhiana were studied. Territorywise incidence was 57.2% - anterior wall MI, 27.2% - inferior wall MI, and 4.16% - lateral wall MI. Survival in

cardiac failure in the presence of LBBB was improved by temporary pacing. Complete heart block was more common in inferior wall MI (18.05%) than in anterior wall MI (8.2%). Mortality was 15.8% and 50% respectively[26].

In a study by Jones M E et al[5] in 1976,556 patients with acute MI admitted to CCU at Aberden Royal Infermary were studied. Incidence of conduction disturbances was 34.9% (194 patients). Complete LBBB was observed in 23 patients and carried 61% mortality. Complete RBBB was seen in 8 patients with a mortality of 38% . LAHB was observed in 72 patients with 13% mortality. Left posterior hemiblock was observed in 32 patients with 19% mortality. Bifascicular block was observed in 59 patients with a mortality of 52%. Complete atrioventricular block was seen in 51 patient with a mortality of 87%[27].

In a study by Patricia Jabre et al, 3220 patients hospitalized with incident MI from 1983 to 2007 in Olmsted country were studied. Atrial fibrillation was identified in these patients by diagnostic codes and ECG. 304 patients had AF before MI. 729 patients developed AF after MI (218 within 2 days, 119 between 3 and 30 days, and 392 more than 30 days post MI). Mortality risk was increased in patients with AF irrespective of clinical characteristics and heart failure. Mortality risk varied according to the timing of AF after MI, with maximum risk for MI occurring 30 days post MI[28].

Martin St.John Sutton et al studied 263 subjects in whom Transthoracic 2D Echocardiogram and arrhythmia monitoring were performed at baseline , 1 year and 2 years after MI. ECG was assessed for the prevalence of VT and VPC. The study showed the prevalence of VT and PVC's in 20% and 29% at baseline, 22% and 35% at 1 year, 33% and 39% at 2 years respectively. This study concluded that alteration in

LV architecture and function by post MI remodelling makes it a substrate for ventricular arrhythmias[29].

Jane S.Saczynski et al studied 7513 residents of the Worcester, Massachusetts,metropolitan area, and found that the overall incidence of AF complicating AMI was 13.3%. Mortality was high in patients who developed AF. They concluded that AF remains a frequent complication of AMI and it has poor prognosis[30].

Keith.H.Newby et all did a huge study, which was conducted on 40,895 patients with Ventricular Arrhythmia. 4,188(10.2%) had sustained Ventricular Tachycardia , Ventricular fibrillation or both. Occurrence of sustained Ventricular Tachycardia or Ventricular Fibrillation, whether late or early, has a negative impact on patient outcome. Mortality is high in Patients with both Ventricular Tachycardia and Ventricular fibrillation[31].

In a study conducted by Tom P Aufderheide, 90 % of acute MI patients have some cardiac rhythm abnormality &cardiac conduction disturbance was seen in 25% of patients. These abnormalities were recorded within 24 hrs of infarct onset. Incidence of VF was high in the first hour following MI (4.5%). All MI patients have increased ANS activity, which results in sinus bradycardia, atrioventricular block[32].

In a study by Jonathan P.Piccini et all they concluded that of the 9015 patients who underwent percutaneous coronary intervention for Acute MI, sustained ventricular tachycardia or fibrillation before revascularization developed in 472 (5.2%) patients. Cardiogenic shock was an independent predictor of sustained VT/VF. Greater mortality was seen in patients with sustained VT/VF[33].

In a study conducted by Mohit J Shah, Nikita R. Bhatt, Ajay Dabhi, P.B. Thorat, KetanChudasama, JigarPatel - 100 cases of acute MI were studied. Males (70%) had higher incidence of MI than females (30%). Incidence of Anterior wall infarcts (69%) was higher than inferior wall (26%). Incidence of VPC (36.23%) was the highest in anterior wall MI. Incidence of complete heart block (26.9%) was the highest in inferior wall MI. A large number of arrhythmias were terminated pharmacologically (39%) whereas 13 % of the arrhythmias persisted in spite of treatment[34].

Haitham Hreybe MD, Samir Saba MD did a huge study on patients with a primary diagnosis of AMI from 1996 – 2003, in which they included 21,807 patients, representing 2,632,217 hospital discharges in the United States. Complete heart block was common in inferior wall MI than anterior wall MI (3.7% vs 1.0%, hazard ratio [HR]=3.9, $p<0.001$), but these patients are less likely to die prior to hospital discharge (7.7% vs 11.3%, HR=0.65, $p<0.001$). Conduction disturbances are more likely to develop in patients with an inferior or posterior AMI when compared to patients with an anterior or lateral AMI. But , anterior or lateral MI is a significant predictor of in-hospital death[35].

In a study by Joern Schmitt, Gabor Duray, Bernard J. Gersh, and Stefan H. Hohnloser,they concluded that atrial fibrillation (AF), is the most common arrhythmia complicating AMI (6 – 21 %). Advancing age, heart failure, depressed LV function are the predictors of AMI. Evidence demonstrates that AF in hospitalized acute MI patients affect the prognosis adversely. This apply for all patient populations studied excluding the differences related to the treatment of AMI (i.e. no reperfusion therapy vs. thrombolysis vs. percutaneous coronary intervention). Patients who have

congestive heart failure and/or a reduced left ventricular ejection fraction have high mortality. AF complicating AMI not only increases the risk for ischaemic stroke during hospitalization but also during follow-up. Transient AF which has reversed back to sinus rhythm at the time of discharge also carries same risk . Prospective studies are needed to evaluate optimal therapeutic approaches for AMI patients complicated by AF[36].

G. E. Honey and S. C. Truelove showed in their study that death occurs commonly during the first 24 hours after infarction, and that the risk of death falls after 48 hours, and that the chances of survival are high if the patient gets through the first week safely. The percentage of mortality for patients admitted to hospital after acute myocardial infarction lies between 30 and 40%. But many reach a hospital bed after the most critical 24 hours have already passed. If the patients are admitted earlier and continuous electrocardiographic monitoring is done, the incidence of recorded arrhythmias will be higher.

Dr. E. Stock and his colleagues did a study in the coronary care unit at the Royal Melbourne Hospital, and tried to elucidate the influence of particular arrhythmias on mortality. The development of an ectopic rhythm or disturbance in conduction occurring after AMI leads to grave deterioration in cardiac function. Supraventricular tachycardia, flutter, or fibrillation shortens the diastole , which leads to reduction of ventricular filling in diastole and further impairs blood flow in the coronary arteries. In 20 % of patients sinus or nodal bradycardia occurs at some time, which leads to reduction in the maximal stroke volume. Sometimes atrioventricular block develops,

which results in an even more profound fall in output . The loss of atrial function in nodal rhythms or heart block causes further fall in stroke volume, and hence output.

Also slow heart is an unstable heart which can harbinge lot of arrhythmias, especially when the rate is in 50s. Ectopic beats frequently presage major arrhythmias, especially when they fall on the T wave of the preceding beat. This phenomenon which is called “R on T” is the major predisposing factor for ventricular arrhythmias. When ventricular fibrillation occurs, effective cardiac contraction ceases. The application of external cardiac massage and electrical countershock has saved the lives of many patients in hospital.

The incidence of major arrhythmias and conduction defects is high even in cases of clinically mild infarction. This finding emphasizes the need to admit all patients with a recent infarct to CCU, however well they may appear initially. Among the patients at Melbourne with severe infarction 27 out of 47 patients with major or multiple arrhythmias died, however in patients who had minor or no arrhythmias only 14 out of 46 patients died. Also the incidence of major and of multiple arrhythmias rose in proportion to the severity of infarction. When the infarct is complicated by left ventricular failure or a low output and hypotesion, major and recurrent arrhythmias are common. This is because of the resulting derangement in the milieu interior of the heart. When the cardiac output falls ,it is associated with an increased release of adrenaline and noradrenaline, which tend to maintain the blood pressure by causing vasoconstriction but which also increase cardiac irritability. As a result there is accumulation of anaerobic metabolites, acidosis, and further myocardial depression.

Hypoxemia and acidosis are not related to the arrhythmias occurring in patients with minor infarcts. Lignocaine has a role in preventing ventricular ectopic beats and the recurrence of ventricular tachycardia or fibrillation after successful electricalreversion. Digitalis is indicated in tachyarrhythmias occurring in the setting of left ventricular failure and is the drug of choice for the treatment of recurrent supraventricular arrhythmias. Atropine is the drug of choice for sinus or nodal bradycardia, however immediate insertion of endocardial pacing catheter is needed in case of atrioventricular block. Fall in cardiac output and the emergence of serious ectopic escape rhythms is prevented by early pacing.

Ronald W F Campbell, Alan Murray, Desmond G Julian studied the prevalence of ventricular arrhythmias in the first 12 hours following acute myocardial infarction. They compared 17 patients who developed primary ventricular fibrillation and 21 apparently similar patients without primary ventricular fibrillation. This study showed that different ventricular arrhythmias occur at different rate in acute myocardial infarction. Primary ventricular fibrillation was closely associated with R-on-T ventricular ectopic complexes[37].

Nagabhushana S, Ranjith kumar GK, Ranganatha M, Virupakshappa – studied 100 patients of Acute Myocardial Infarction (AMI) admitted to ICCU of Mc Gann Hospital from April 2015 to June 2015. They recorded ECGs immediately after admission, then four hourly till the hospital stay and whenever required. Cardiac enzymes and 2D echocardiography were done to confirm MI. The mean age as per their observation was 52.91 years, with male to female ratio being 2.9:1. Incidence of arrhythmias in their study was 89%, with a majority (67%) of arrhythmias occurring

in the first 24 hours. Sinus tachycardia (48%), ventricular premature beats (VPCs) (24%), Sinus bradycardia (22%) and atrial premature complexes (15%) were the commoner arrhythmias. Atrioventricular blocks (93.4%) were the commonest arrhythmia occurring in inferior wall infarctions. Incidence of arrhythmias was 90.91% in anterior wall MIs, incidence of arrhythmias was 83.33% in inferior wall MIs[38].

Usefulness of the accerelated idioventricular rhythm as a marker for myocardial necrosis and reperfusion during thrombolytic therapy in acute myocardial infarction was studied by GorgelsAP, Vos MA ,LetschIS, Verschuuren EA, Bar FW, Janssen JH, Wellens HJ. They concluded that reperfusion of left anterior descending fascicle shows most configurations of accelerated idioventricular rhythm with QRS width whereas reperfusion of circumflex branch never had a left bundle branch like configuration. AIVR occurring during persistent ischemic pain can be used as a marker of myocardial necrosis as well as reperfusion of infarcted vessel[39].

Van der Weg K , Majidi M ,HaeckJD,TijssenJG,Green CL, Koch KT , Krucoff MW, GorgelsAP , de Winter RJ did a study to determine whether Ventricular arrhythmia can be used as an independent indicator of larger infarct size even in optimal reperfusion in STEMI. They concluded that in those with optimal epicardial and microvascular reperfusion ,Ventricular arrhythmia bursts were associated with larger infarct size[40].

BassanR , Mala IG , Bozza A, Amino JG , Santos M did a study to determine whether Atrioventricular block in acute inferior wall myocardial infarction is associated with obstruction of left anterior descending coronary artery. They observed

that patients presenting with inferior myocardial infarction and left anterior descending artery obstruction have increased risk of developing heart block in the acute phase of infarction which also explains the observations that the proximal AV conduction system has a dual arterial blood supply from right &left anterior descending coronary arteries[41].

Six AJ ,Louwerenburg JH , Kingma JH , Robies de Medina EO , Van Hemel NM did a study to assess the predictive value of ventricular arrhythmia for patency of the infarct related coronary artery after thrombolytic therapy. Their sudy revealed that 81% of the patients with accelerated idioventricular rhythm or non sustained ventricular tachycardia or both after thrombolysis had a patent infarct related vessel[42].

Majidi M , Kosinski AS , Al-Khatib SM , Lemmert ME , Smolders L, vanWeertA, Reiber JH, Tzivoni D, Bar FW, Wellens HJ, Gorgels AP, Krucoff M did a study to assess whether Reperfusion ventricular arrhythmia bursts predict larger infarct size despite TIMI 3 flow restoration with primary angioplasty for anterior ST Elevation myocardial infarction. They have concluded that successful epicardial reperfusion with primary percutaneous coronary intervention for ST elevation myocardial infarction can paradoxically evoke myocardial reperfusion injury signalled by ventricular arrhythmias[43].

Gorenek B, Dogan V, Yasar B, Birdane A, Cavusoglu Y, UnalirA, Ata N, Timuralp B have studied the Initiation patterns of monomorphic ventricular tachycardia in acute myocardial infarction by analysing the rhythm strips. From the analysis of 255 rhythm strips ,it is seen that Monomorphic Ventricular Tachycardia is

most often preceded by ventricular ectopic beats in the acute phase of Myocardial Infarction[44]

Smith PJ, Blumenthal JA, Babyak MA, Georgiades A, Sherwood A, Sketch MH Jr, Watkins LL studied the impact of self reported stress after myocardial infarction. Temporal analyses of the relationship between stress &ectopy showed that psychological stress predicts increased arrhythmic activity during routine daily activities in post MI patients[45].

Talbot S studied the prognostic importance of ventricular extrasystoles in acute myocardial infarction. He observed that three quarters of the severe arrhythmias occurred in the first 24 hours following acute myocardial infarction and 60% of these were preceded by either multiform ventricular extrasystoles or extrasystoles with variable coupling[46].

Khan A, Nadeem S, Kokane H, Thummar A, Lokhandwala Y, Mahajan AU, Nathani PJ did a study to assess whether accelerated idioventricular rhythm is a good marker for reperfusion after streptokinase. They concluded that Accelerated Idioventricular Rhythm is a common arrhythmia in patients with ST segment elevation myocardial infarction. Early AIVR can be used as an additive criterion to ST segment resolution as a non invasive marker of successful thrombolysis with streptokinase[47].

Iakovlev VA, Tarasov VA studied the chronostructure of ectopic heart activity in the acute phase of myocardial infarction and found that the probability of ventricular arrhythmia is maximal in transmural MI and the most arrhythmogenic period being the first 3 days of transmural MI. Ventricular ectopic activity enhance in

the morning , afternoon and on the first 5 days after midnight. Atrial ectopic activity in transmural MI significantly enhance in the morning ,afternoon and at night[48].

Chiladakis JA, PashalisA,Patsouras N, Manolis AS studied the autonomic patterns preceding and following accelerated idioventricular rhythm in acute myocardial infarction. They concluded that reperfusion induced accelerated idioventricular arrhythmias are modulated by sympathetic stimulatory effects and the conterregulatory vagal response exert a profound effect upon its suppression[49].

Suguira T, Iwasaka T, Koito H, Kimura Y, Inada M, Spodick DH studied the supraventricular arrhythmias in the late hospital phase of acute Q wave myocardial infarction . According to the study conducted, predictors of supraventricular tachycardias were moist rales, digitalis, age,and cardiothoracic ratio. Aging , hemodynamic change imposed on the left ventricle and arrhythmic effects of digitalis are the major factors associated with supraventricular arrhythmias in the late hospital phase of acute MI[50].

Sasikumar N, Kuladhipati I studied the possibility of spontaneous recovery of complete atrioventricular block complicating acute anterior wall ST elevation myocardial infarction. Complete AV block is one of the worst prognostic indicators following acute myocardial infarction. However, Complete AV block produced by reocclusion of an infarct related artery can be reversed by percutaneous coronary angioplasty of the infarct related artery[51].

Ricci JM, Dukkipati SR, Pica MC, Haines DE, Goldstein JA studied malignant ventricular arrhythmias in patients with acute right ventricular infarction undergoing mechanical reperfusion. This Study concluded that Malignant Ventricular

Arrhythmias are common in right ventricular infarctions which commonly occur before perfusion and are associated with larger infarcts[52].

Fiol Sala M, Marrugat J, Bergada Garcia J, GuindoSoldevila J, Bayes de Luna A studied the differential characteristics of early ventricular arrhyhmias following a myocardial infarct in patients with and without ventricular fibrillation. This study showed that R on T phenomenon or short prematurity index and fast runs of ventricular tachycardia along with other parameters such as inferior site of infarct, sum of ST -3 leads > 10 mm and basic heart rate > 100 beats per minute are the characteristic ventricular arrhythmias preceding ventricular fibrillation episodes[53].

Araszkiewicz A, Grygier M, Pyda M, Rajewska J, Lesiak M, Grajek S did a study to assess whether postconditioning attenuates early ventricular arrhythmias in patients with high risk ST segment elevation myocardial infarction. The study demonstrated that postconditioning may reduce the occurrence of malignant ventricular arrhythmias in patients with STEMI treated with primary PCI[54].

Cricri P, Trachsel LD, Muller P, Wackerlin A, Reinhart WH, Bonetti PO studied the Incidence and time frame of life threatening arrhythmias in patients undergoing primary percutaneous coronary intervention. They found that most of the life threatening arrhythmias occur during the primary percutaneous coronary intervention procedure. Post procedural life threatening arrhythmias are limited to the first 24 hours after PPCI[55].

JurkovicovaO ,Cagan S studied the supraventricular arrhythmias and disorders of atrioventricular and intraventricular conduction defects in patients with acute myocardial infarction.Factors associated with the development of supraventricular

arrhythmias are Atrial dilatation ,increase in intra atrial pressure ,autonomic disturbances, acidosis , global hypoxia. SVA complicate the course of inferior , posterior and lateral myocardial infarction. But also occurs in right ventricular myocardial infarction and pericarditis. AV block occurring in inferior myocardial infarction is frequently reversible whereas that occurring in anterior myocardial infarction is persistent and irreversible. Bundle branch block can sometimes be used as a marker of multivessel disease[56].

Gore JM , Ball SP, Corrao JM, Goldberg RJ did a study to assess arrhythmias as a marker of coronary artery reperfusion following thrombolytic therapy. Conclusion of the study showed that arrhythmias in general should not be used as markers for coronary reperfusion. Only bradyarrhythmias can be used as a marker of reperfusion of right coronary artery[57].

BaroidSS ,Herweg B did a study in the name of second degree atrioventricular block revisited. Type II AV block appears to be all or none conduction without visible changes in the AV time before and after the blocked impulse. Also absence of sinus slowing is an important criterion of type II AV block due to vagal surge causing simultaneous sinus slowing and AV nodal block. Diagnosis of type II AV block can only be made if the first postblock P wave is followed by shortened PR interval or the P wave is not discernible[58].

DurakI ,Kudaiberdieva G, Gorenek B studied the prognostic implications of arrhythmias during primary percutaneous coronary interventions for ST elevation myocardial infarction. They concluded that sustained ventricular arrhythmias developing during or early after PCI and reperfusion of the blocked vessel do not

affect the prognosis.Ventricular arrhythmias which are associated with incomplete revascularistion and ongoing ischemia, new onset atrial fibrillation ,high degree atrioventricular block are associated with poor prognosis[59].

Brenhardt G, SeipelL ,Loogen Fdid a study to assess prognostic significance of arrhythmias in acute myocardial infarction. The study concluded that frequent ectopic beats, multifocal ectopic beats, ventricular bigeminy, ventricular salvoes, ventricular tachycardia and the R on T phenomenon are considered warning arrhythmias before ventricular fibrillation develops following myocardial infarction. Recent studies show that lidocaine can be used to prevent ventriculat fibrillation following MI[60].

Wildi K, Cucult F, Twerenbold R, Marker T, RubiniGimenez M, Reichlin T, Haaf P, Monsch R, Mersch S, Hunziker P, Bingisser R, Osswald S, Erne P, Mueller C studied the incidence and timing of serious arrhythmias after early revascularisation in non ST – elevation myocardial infarction. The study concluded that unlike STEMI ,in NSTEMI the incidence of serious arrhythmias after successful early revascularisation seems to be very low[61].

Comerford TJ, Propert DB studied accelerated idioventricular rhythm in patients without acute myocardial infarction. They concluded that Accelerated Idioventricular Rhythm can be seen in patients with acute myocardial infarction, subarachnoid haemorrhage, digitalis excess, and in those with rheumatic , primary myocardial ,and hypertensive heart disease. Study shows that AIVR occurs infrequently in patients without demonstrable heart disease[62].

Jurkovicova O, Cagan s did a study in the name of Reperfusion arrhythmias. Reperfusion arrhythmias is considered an early (within 6 hours of thrombolysis),

frequent (> 30 episodes/hour) ,and repetitive (occurring during more than 3 consecutive hours) accelerated idioventricular arrhythmias. This study found that Reperfusion arrhythmias is an important non invasive marker of coronary artery recanalization. Arrhythmias such as frequent premature ventricular complexes, increase in episodes of nonsustained ventricular tachycardia, sinus bradycardia, high degree atrioventricular blocks are also considered as markers of reperfusion[63].

Tatli E, Alicik G, Buturak A, Yilmaztepe M, Aktoz M studied arrhythmias following revascularization procedures in the course of acute myocardial infarction to assess whether they are indicators of reperfusion or ongoing ischemia. Study suggests that ongoing vascular occlusion and ischemia may lead to arrhythmias which can not be distinguished from reperfusion arrhythmias[64].

Ilia R, Amit G, Cafri C, Gilutz H, Abu-Ful A, Weinstein JM, Yaroslavtsev S, Gueron M, Zahger D did a study to assess whether reperfusion arrhythmias occured during coronary angioplasty for acute myocardial infarction predict ST – segment resolution. They concluded that Reperfusion arrhythmias following coronary angioplasty for acute myocardial infarction are highly specific marker for ST resolution and also indicates successful microvascular reperfusion[65].

Gao R studied Reperfusion arrhythmias in acute myocardial infarction. This study concluded that Reperfusion arrhythmias following acute myocardial infarction remains an indicator of recanalization of infarcted coronary artery.Also these arrhythmias are treated by anti arrhythmic agents, electric defibrillation, but could not be prevented by lidocaine[66].

Maclellan- Tobert SG, Porter CJ studied whether Accelerated idioventricular rhythm is a benign arrhythmia in childhood. This study showed that AIVR seems to be a benign arrhythmia in childhood. But complete resolution of the arrhythmia doesn't occur. Also treatment was not effective in controlling the arrhythmia and is considered unnecessary[67].

Hoffman I, Zolnick MR, Bunn C studied Transient post reperfusion left bundle branch block and accelerated idioventricular rhythm with paradoxical QRS narrowing. They concluded that AIVR commonly follows coronary reperfusion whereas transient left bundle branch block is only rarely seen after interventional reperfusion. Study reports post perfusion AIVR and a simultaneous transient LBBB with fusion complexes cause paradoxical QRS narrowing[68].

Henriques JP, Gheeraert PJ, OttervangerJP, de Boer MJ, Dambrink JH, Gosselink AT, van't Hof AW, Hoorntje JC, Suryapranata H, Zijlstra F studied the characteristics of Ventricular fibrillation in acute myocardial infarction before and during primary PCI. Study shows that patients with early ventricular fibrillation before reperfusion have characteristics which is different from the ventricular fibrillation occurring after reperfusion. Also the Timing of ventricular fibrillation is determined by infarct location[69].

Osmancik PP, Stros P, Herman D studied the In – hospital arrhythmias in patients with acute myocardial infarction and their relation to the reperfusion strategy and their prognostic impact. The presence of AIVR along with non invasive markers like ST segment resolution is connected with high probability of successful

reperfusion. Early and successful reperfusion therapy is the best anti- arrhythmia therapeutic measure in patients with myocardial infarction[70].

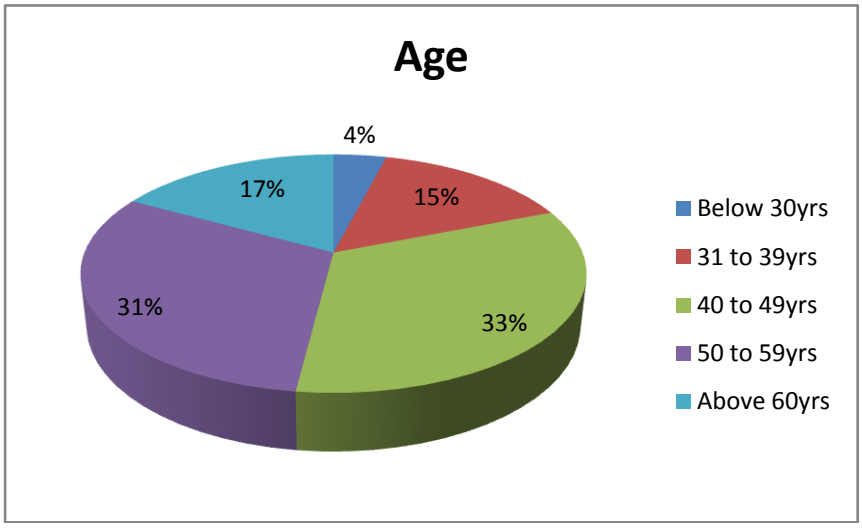
Terkelssen CJ, Sorensen JT, Kaltoft AK, Nielsen SS, Thuesen L, Betker HE, Lassen JF analysed the prevalence and significance of accelerated idioventricular rhythm in patients with ST-elevation myocardial infarction treated with primary percutaneous coronary intervention. They concluded that AIVR is the most frequent arrhythmia occurring during primary percutaneous coronary intervention in patients with ST- elevation myocardial infarction. But it cannot be taken as a marker of successful reperfusion , also AIVR is associated with extensive myocardial damage and delayed microvascular reperfusion[71].

Piccini JP, Berger JS, Brown DL studied early sustained ventricular arrhythmias complicating acute myocardial infarction. The study concluded that sustained ventricular tachycardia /ventricular fibrillation remains a significant complication in patients undergoing percutaneous coronary intervention for acute MI and is associated with high in- hospital mortality[72].

STATISTICS

TABLE -1 AGE DISTRIBUTION

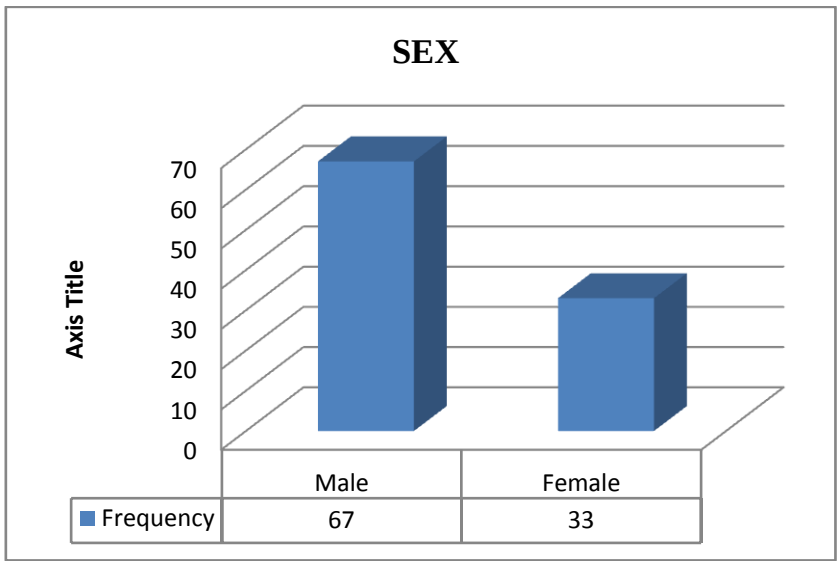
Particulars	Frequency	Percentage
Below 30yrs	4	4.0
31 to 39yrs	15	15.0
40 to 49yrs	33	33.0
50 to 59yrs	31	31.0
Above 60yrs	17	17.0
Total	100	100.0



In this study the total number of cases studied were 100. These cases were divided into five age groups. 4 % of cases were in below 30 yrs age group. 15 % were in 31 – 39 yrs age group. A majority of 33 % belong to 40 – 49 yrs age group. 31 % were in 50 – 59 yrs age group.17 % were in above 60 yrs age group.

TABLE - 2 **SEX DISTRIBUTION**

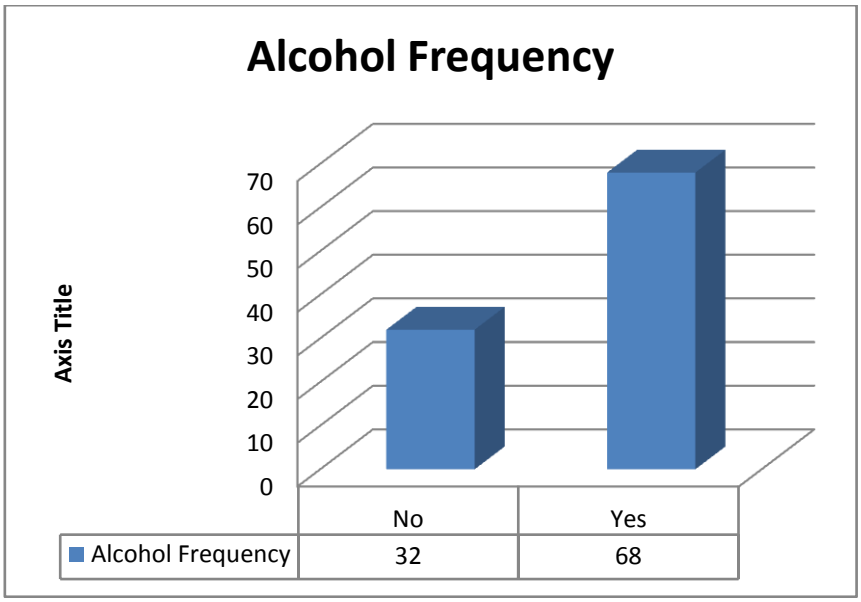
Particulars	Frequency	Percentage
Male	67	67.0
Female	33	33.0
Total	100	100.0



In this study, 67 % of cases were male. 33 % cases were female. Male sex predominance was present in the study group.

TABLE – 3 **ALCOHOL INTAKE DISTRIBUTION**

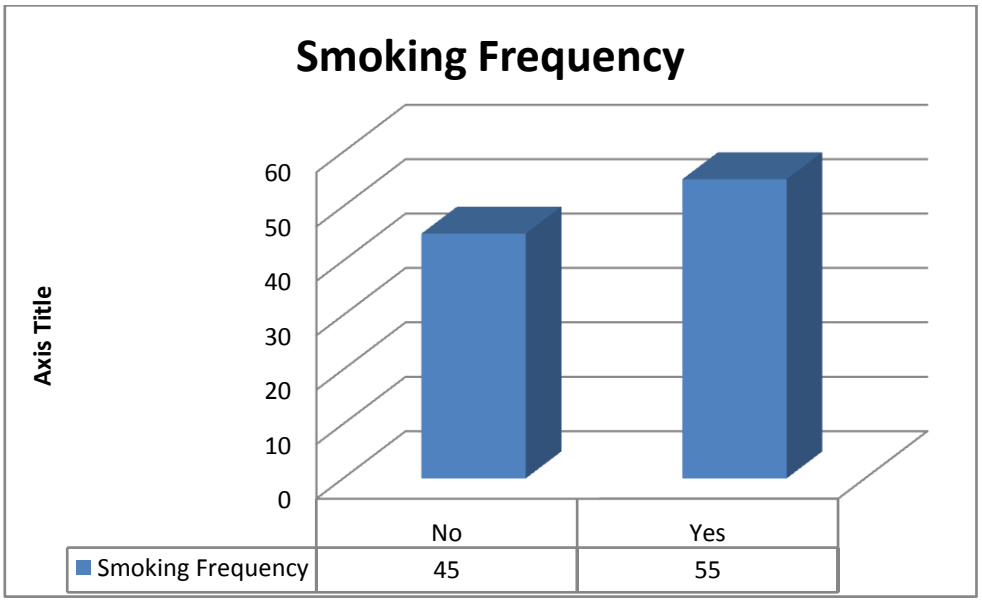
Particulars	Frequency	Percentage
No	32	32.0
Yes	68	68.0
Total	100	100.0



Incidence of alcohol intake in this study population was 68 %. 32 % were non alcoholic. Predominance of alcoholism was present in the study group

TABLE – 4 SMOKING DISTRIBUTION

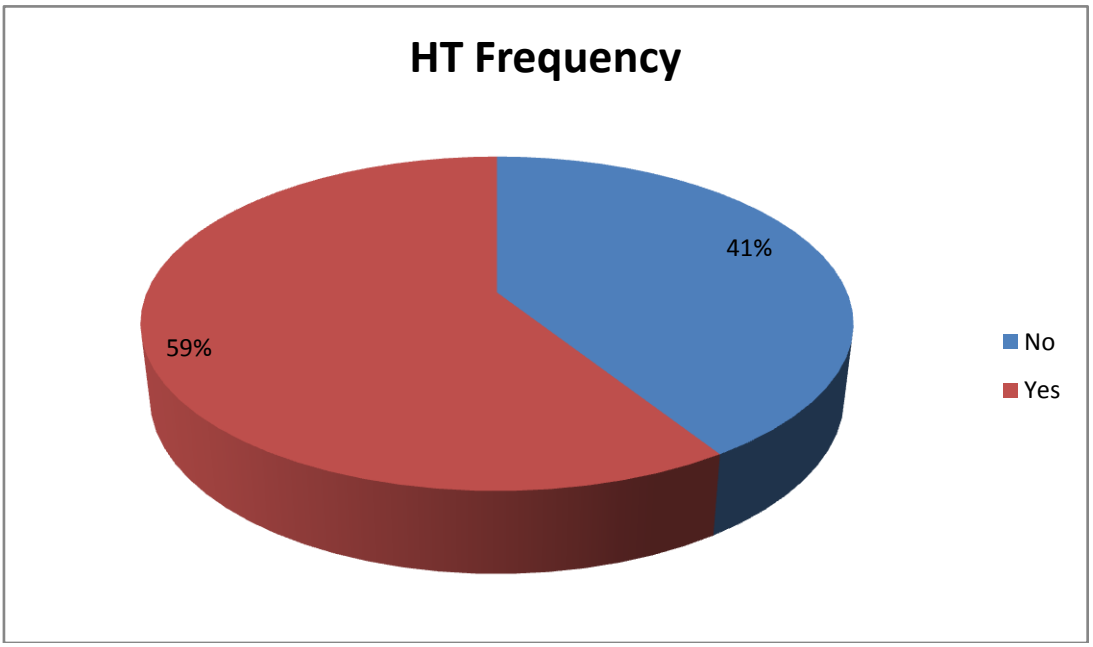
Particulars	Frequency	Percentage
No	45	45.0
Yes	55	55.0
Total	100	100.0



Smoking frequency among the study population was 55 %. 45% of cases were non smokers. Hence this study included predominantly smokers.

TABLE – 5 HYPERTENSION FREQUENCY

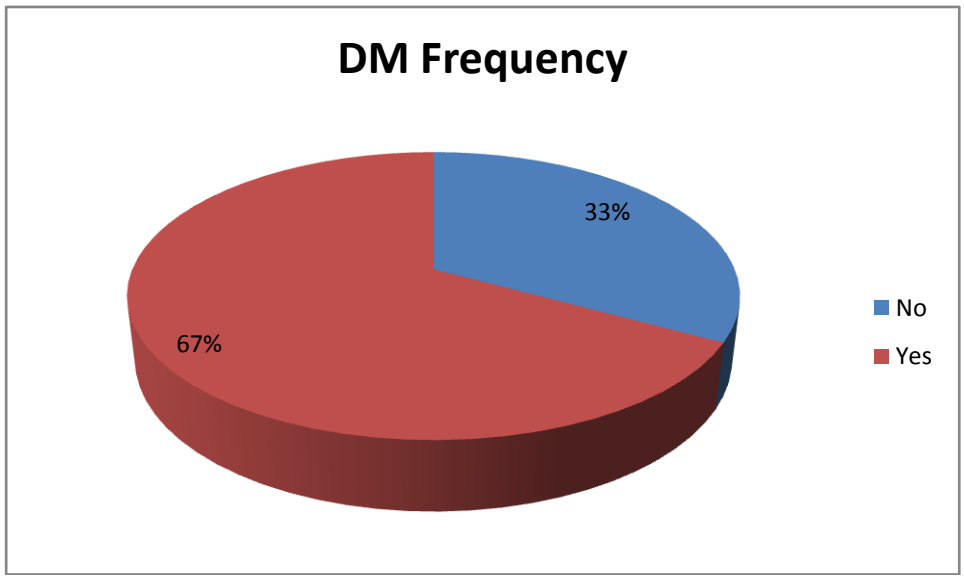
Particulars	Frequency	Percentage
No	41	41.0
Yes	59	59.0
Total	100	100.0



In this study group, 59 % were hypertensives, 41 % were nonhypertensives. This shows predominance of hypertension among the affected people.

TABLE – 6 DIABETES MELLITUS FREQUENCY

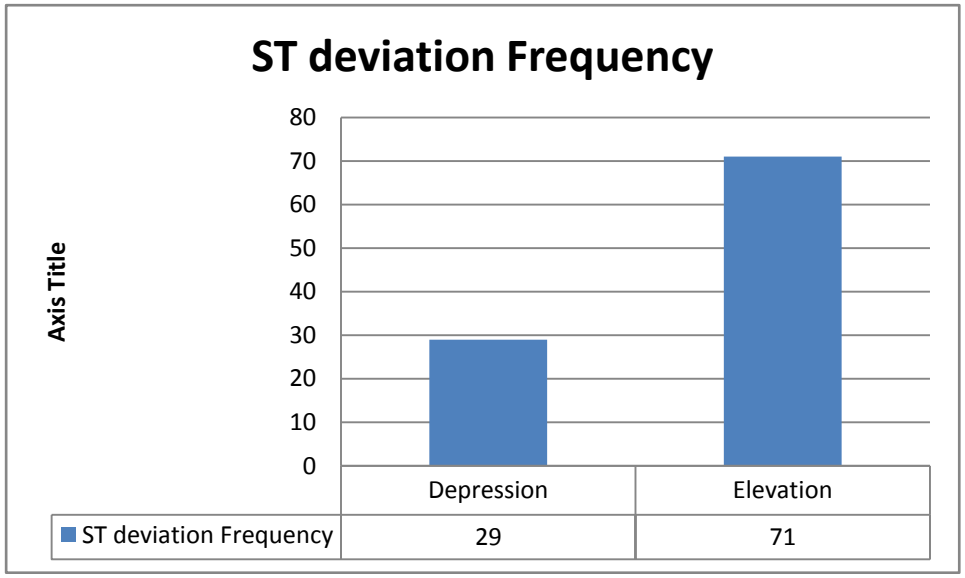
Particulars	Frequency	Percentage
No	33	33.0
Yes	67	67.0
Total	100	100.0



In this study 67 % of cases included were diabetic. 33 % were non diabetic. Predominantly diabetics were the affected population

TABLE – 7 ST DEVIATION DISTRIBUTION

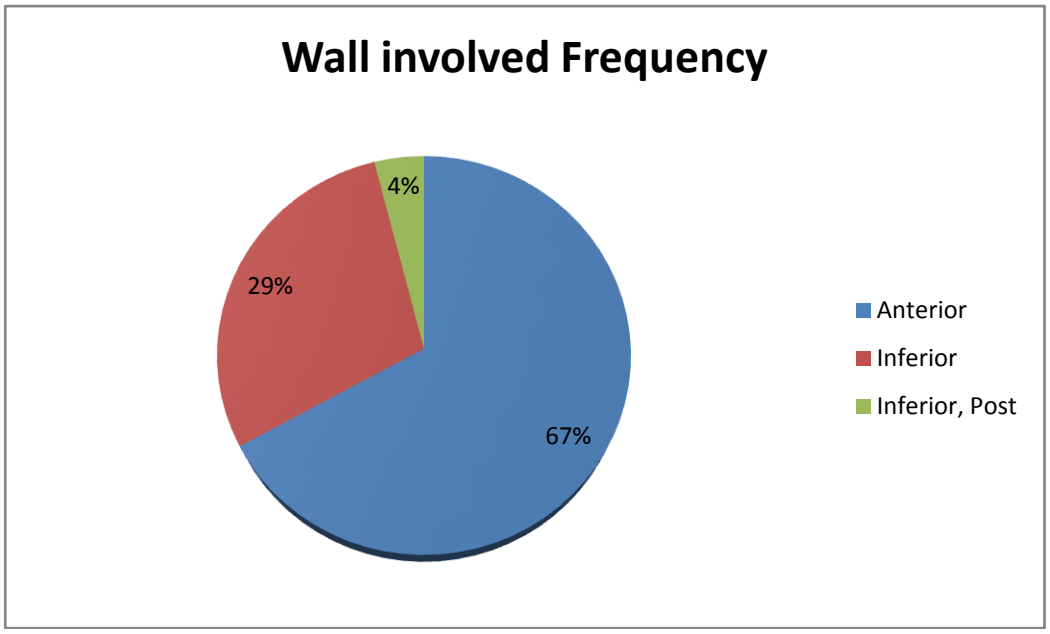
Particulars	Frequency	Percentage
Depression	29	29.0
Elevation	71	71.0
Total	100	100.0



Out of the 100 caes of acute MI studied, 71 % presented with ST elevation. 29 % presented with ST depression. ST elevation MI was the most common presentation

TABLE – 8 **DISTRIBUTION OF WALL OF THE HEART INVOLVED IN ACUTE MI**

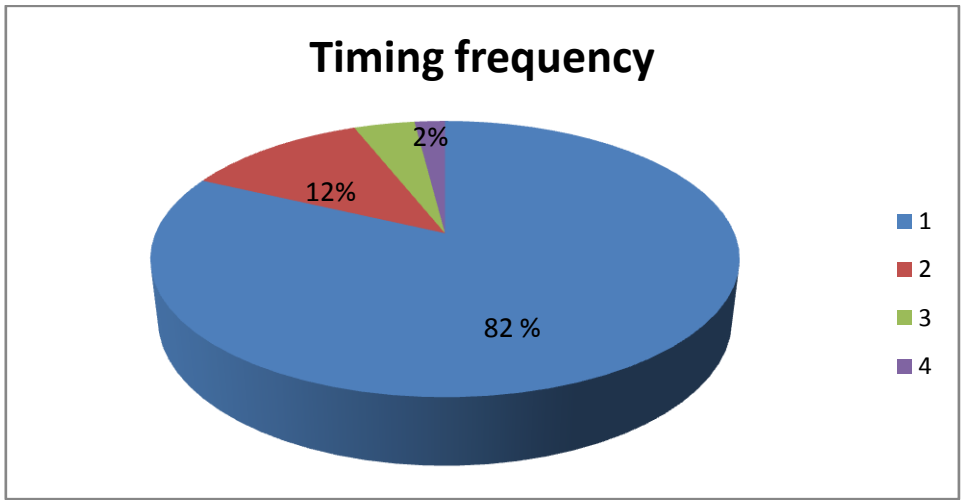
Particulars	Frequency	Percentage
Anterior	67	67.0
Inferior	29	29.0
Inferior, Post	4	4.0
Total	100	100.0



Out of the 100 cases of acute MI studied, 67% had Anterior wall MI, 29 % had inferior wall MI. 4 % had combined involvement of both inferior and posterior wall. Hence anterior wall MI was the commonest, followed by inferior wall MI, and the least was combination of inferior and posterior wall MI.

TABLE 9 TIME DURATION BETWEEN PRESENTATION AND ONSET OF ARRHYTHMIAS IN DAYS

Particulars	Frequency	Percentage
1	82	82.0
2	12	12.0
3	4	4.0
4	2	2.0
Total	100	100.0



Time interval between presentation and onset arrhythmias was studied in the study population. It was found that majority of arrhythmias occurred in the first day post MI which was 82%. 12 % cases developed arrhythmia in the second day. 4 % developed in the third day. Only 2% of cases developed arrhythmia on day 4.

TABLE – 10 DISTRIBUTION OF THE TYPE OF ARRHYTHMIAS

Particulars	Frequency	Percentage
I DEGREE AVB	12	12.0
II DEGREE AVB	9	9.0
AF	4	4.0
AIVR	13	13.0
APC	5	5.0
CHB	6	6.0
LBBB	7	7.0
RBBB	5	5.0
SVT	1	1.0
VF	3	3.0
VPC	27	27.0
VT	8	8.0
Total	100	100.0

12 different types of arrhythmias were observed in the study population. Each occurred at a different frequency. VPC (27%)was clearly the most common arrhythmia observed. Followed by AIVR-13%, I degree AVB-12%, II degree AVB-9%,VT – 8%, LBBB- 7%, CHB – 6%, RBBB, APC each 5%,AF – 4%,VF – 3%, SVT- 1% respectively.

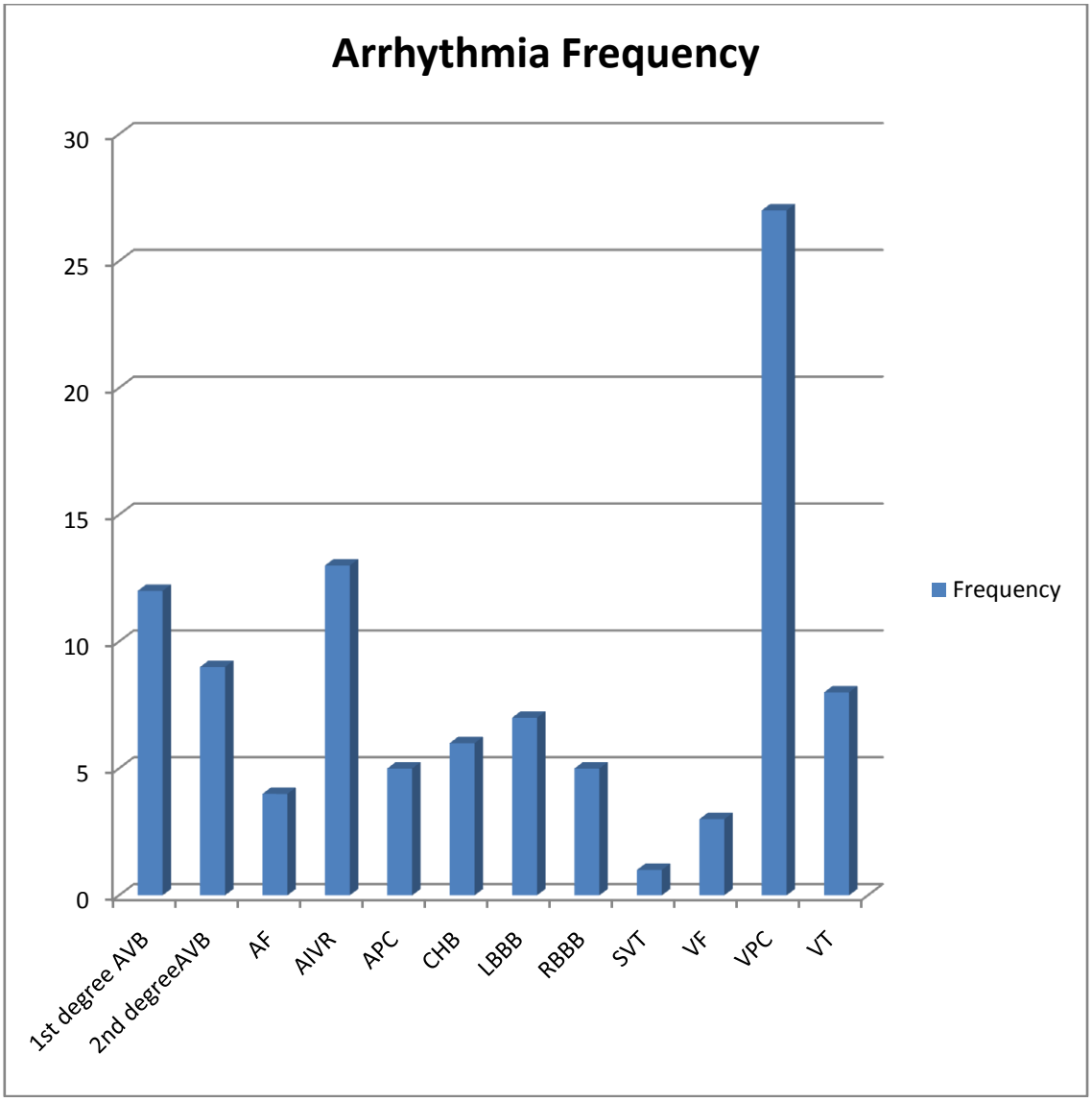


TABLE – 11 DISTRIBUTION OF THE OUTCOME

Particulars	Frequency	Percentage
Dead	18	18.0
Alive	82	82.0
Total	100	100.0

Out of the 100 cases of acute MI studied , 82 % of cases were alive, 18 % of cases died.

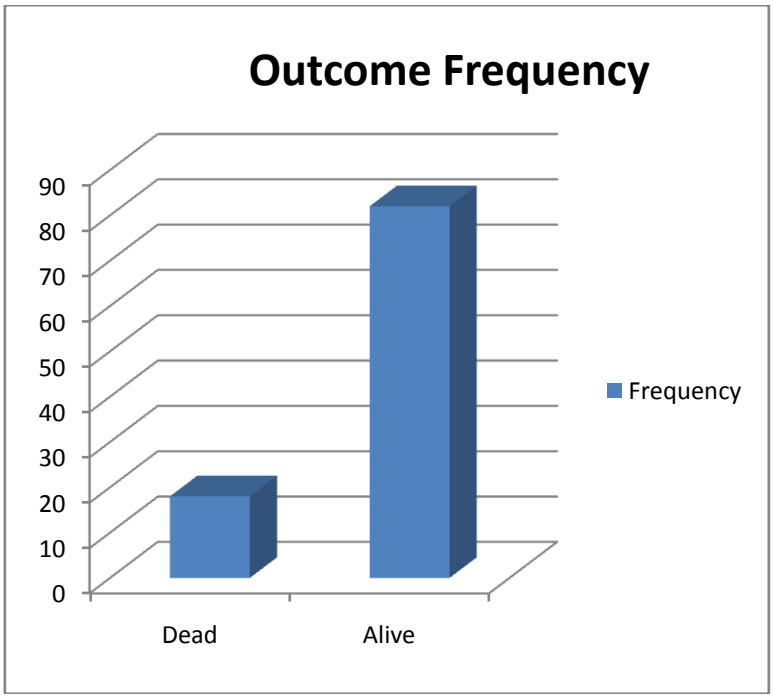
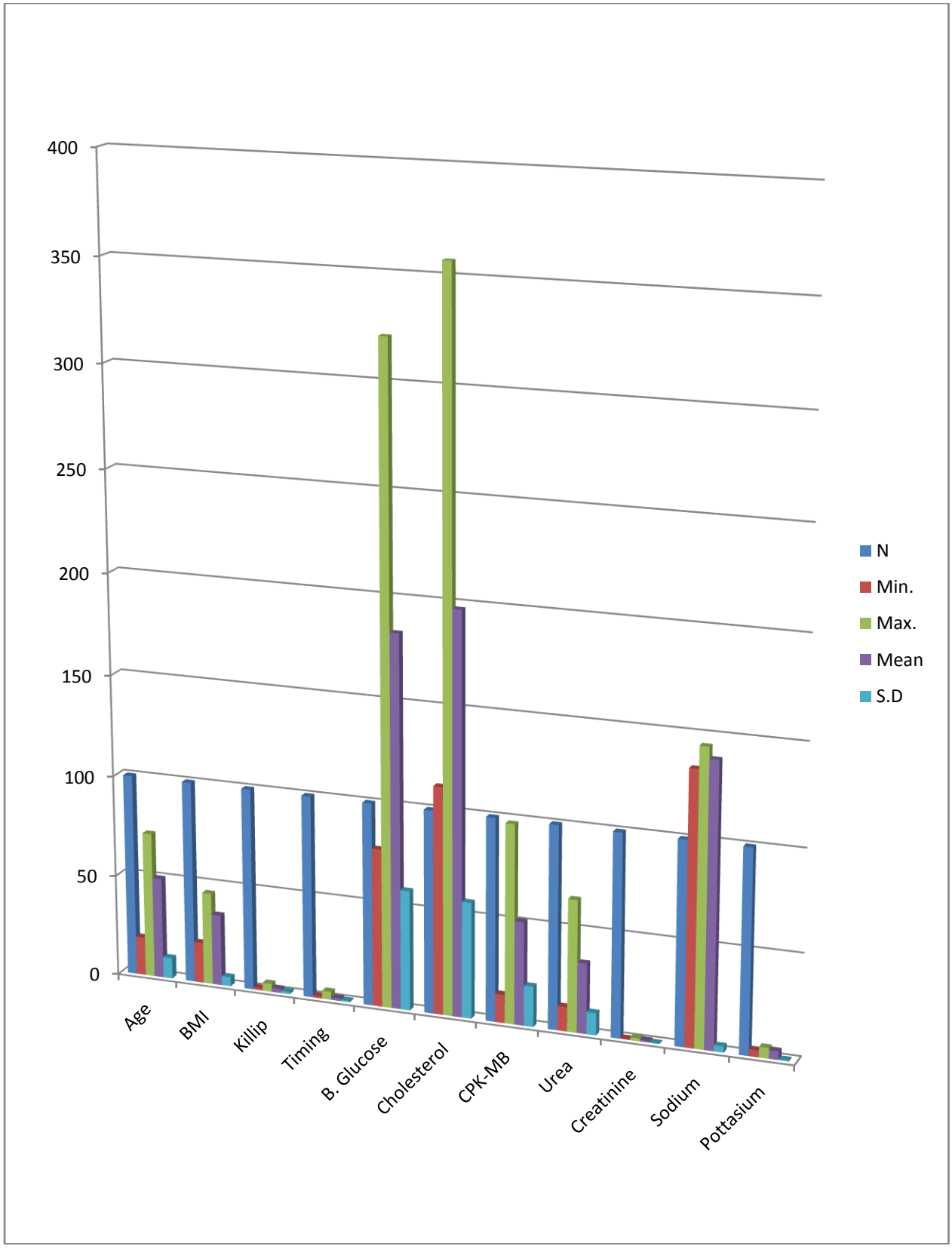


TABLE – 12 DESCRIPTIVE STATISTICS OF AGE, BMI, KILLIP CLASS, TIME INTERVAL , BLOOD GLUCOSE, SERUM CHOLESTEROL, CPK – MB, UREA, CREATININE, SODIUM, POTASSIUM.

Items	N	Min.	Max.	Mean	S.D
Age	100	19	72	49.97	10.318
BMI	100	20.10	45.60	35.2390	4.58039
Killip	100	1	4	2.03	1.123
Timing	100	1	4	1.26	.630
B. Glucose	100	78	321	183.46	59.214
S. Cholesterol	100	112	357	197.49	57.276
CPK-MB	100	14	98	51.02	19.944
Urea	100	12	65	34.58	10.989
Creatinine	100	.40	1.40	.8120	.22351
Sodium	100	134	145	139.27	2.981
Pottasium	100	3.30	5.20	4.2260	.49536


400
350
300
250
200
150
100
50
0
Age
BMI
Killip
Timing
B. Glucose
Cholesterol
CPK-MB
Urea
Creatinine
Sodium
Pottasium
N
Min.
Max.
Mean
S.D

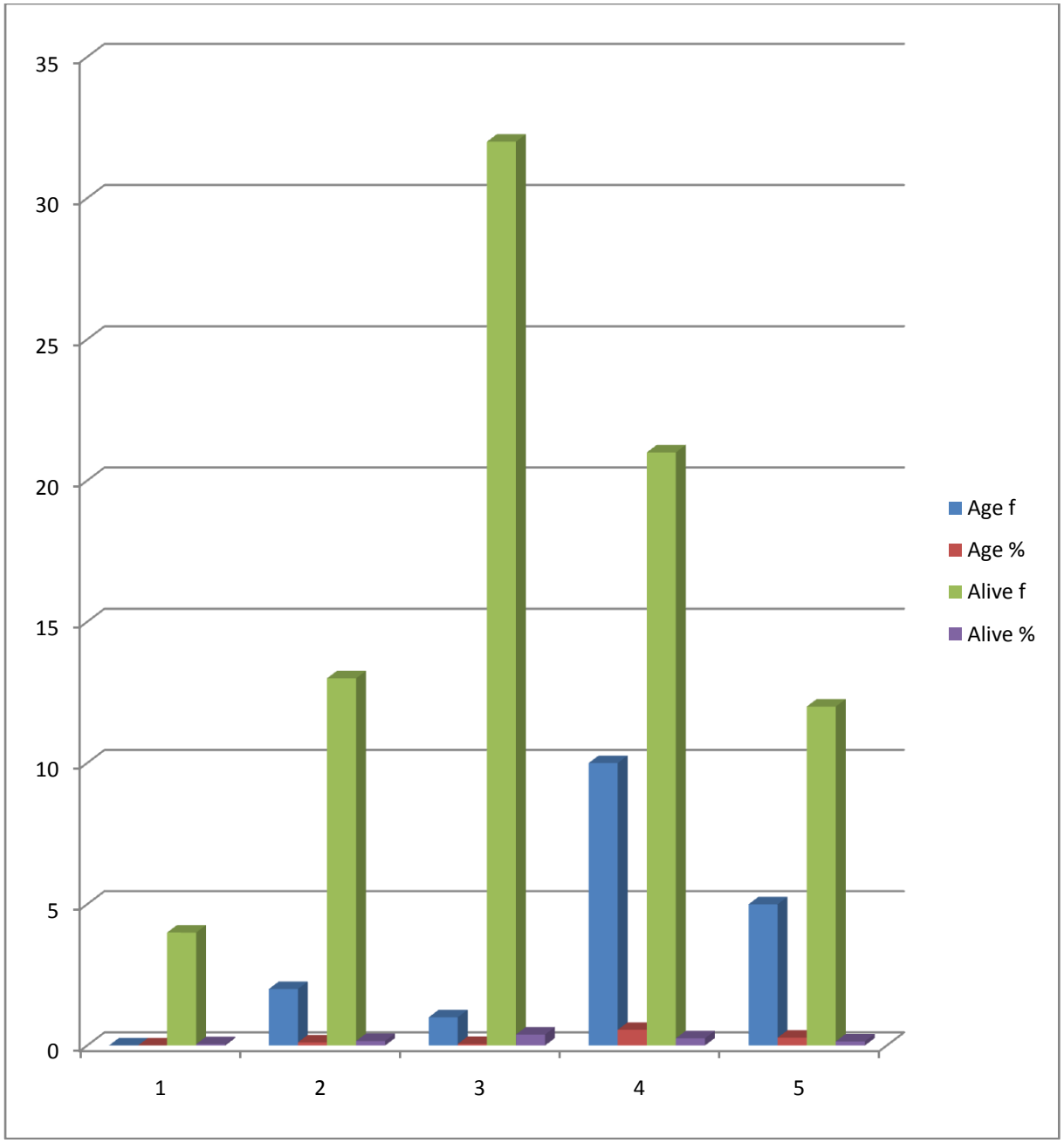
Various parameters like age, BMI, Killip class, time interval behetween the presentation and the onset of arrhythmia, blood glucose, serum cholesterol, CPK – MB, urea, creatinine, sodium, potassium were analysed.

Mean age was 49.97, standard deviation was 10.318. Mean BMI was 35.2390, SD was 4.58039. Mean Killip class was II, SD was 10123. Mean time interval between presentation and onset of arrhythmia was 1.26 days, with a SD of 0.630. Mean B.glucose was 183.46 with a SD of 59.214.

Mean S.cholesterol was 197.49 with a SD of 57.276. Mean CPK-MB was 51.02 with a SD of 19.944. Mean urea was 34.58 with a SD of 10.989. Mean creatinine was 0.8120 with a SD of 0.22351. Mean sodium was 139.27 with a SD of 2.981. Mean potassium was 4.2260 with a SD of 0.49536

TABLE – 13 CHI-SQUARE TEST TO COMPARE THE OUTCOME IN DIFFERENT AGE GROUPS

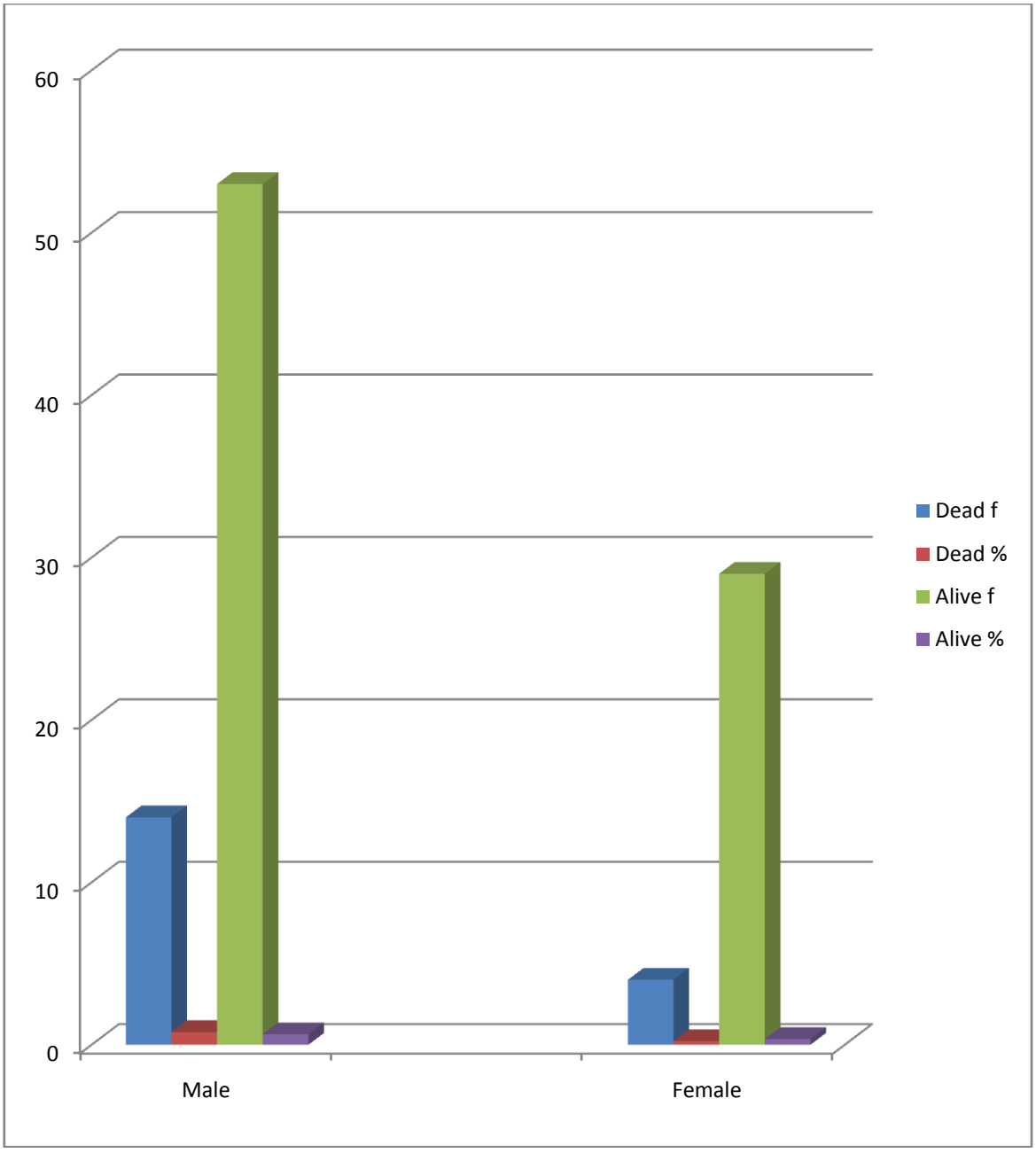
Age	Dead		Alive		Total		Statistical inference
	f	*%*	*f*	*%*	*f*	*%*	
Below 30yrs	0	.0%	4	4.9%	4	4.0%	X^2=11.879 Df=4 .018<0.05 Significant
31 to 39yrs	2	11.1%	13	15.9%	15	15.0%	
40 to 49yrs	1	5.6%	32	39.0%	33	33.0%	
50 to 59yrs	10	55.6%	21	25.6%	31	31.0%	
Above 60yrs	5	27.8%	12	14.6%	17	17.0%	
Total	**18**	**100.0%**	**82**	**100.0%**	**100**	**100.0%**	



Using chi-square test overall outcome is compared with the different age groups of the cases. It showed significant relationship between the age group and the outcome. Death was highest in 50- 59 years age group, and least in below 30 years age group.

TABLE – 14 CHI-SQUARE TEST TO COMPARE OUTCOME IN DIFFERENT SEX GROUPS

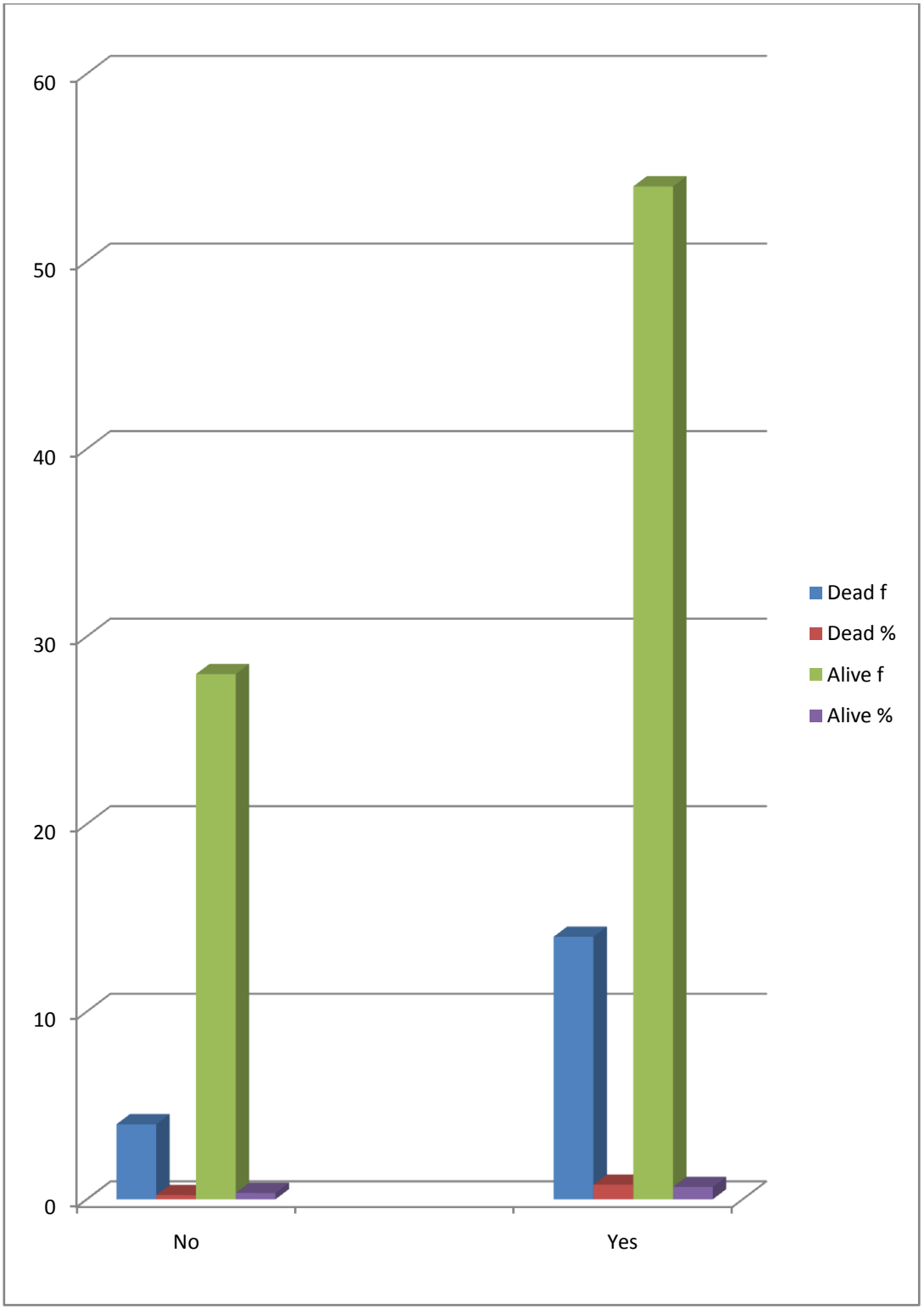
Sex	Dead		Alive		Total		Statistical inference
	f	*%*	*f*	*%*	*f*	*%*	
Male	14	77.8%	53	64.6%	67	67.0%	X^2=1.153 Df=1 .283>0.05 Not Significant
Female	4	22.2%	29	35.4%	33	33.0%	
Total	**18**	**100.0%**	**82**	**100.0%**	**100**	**100.0%**	



Chi – square test was used to compare overall outcome in different sex groups. It was found that sex had no significant effect on mortality in this study.

TABLE – 15 CHI-SQUARE TEST TO COMPARE OUTCOME WITH ALCOHOL INTAKE

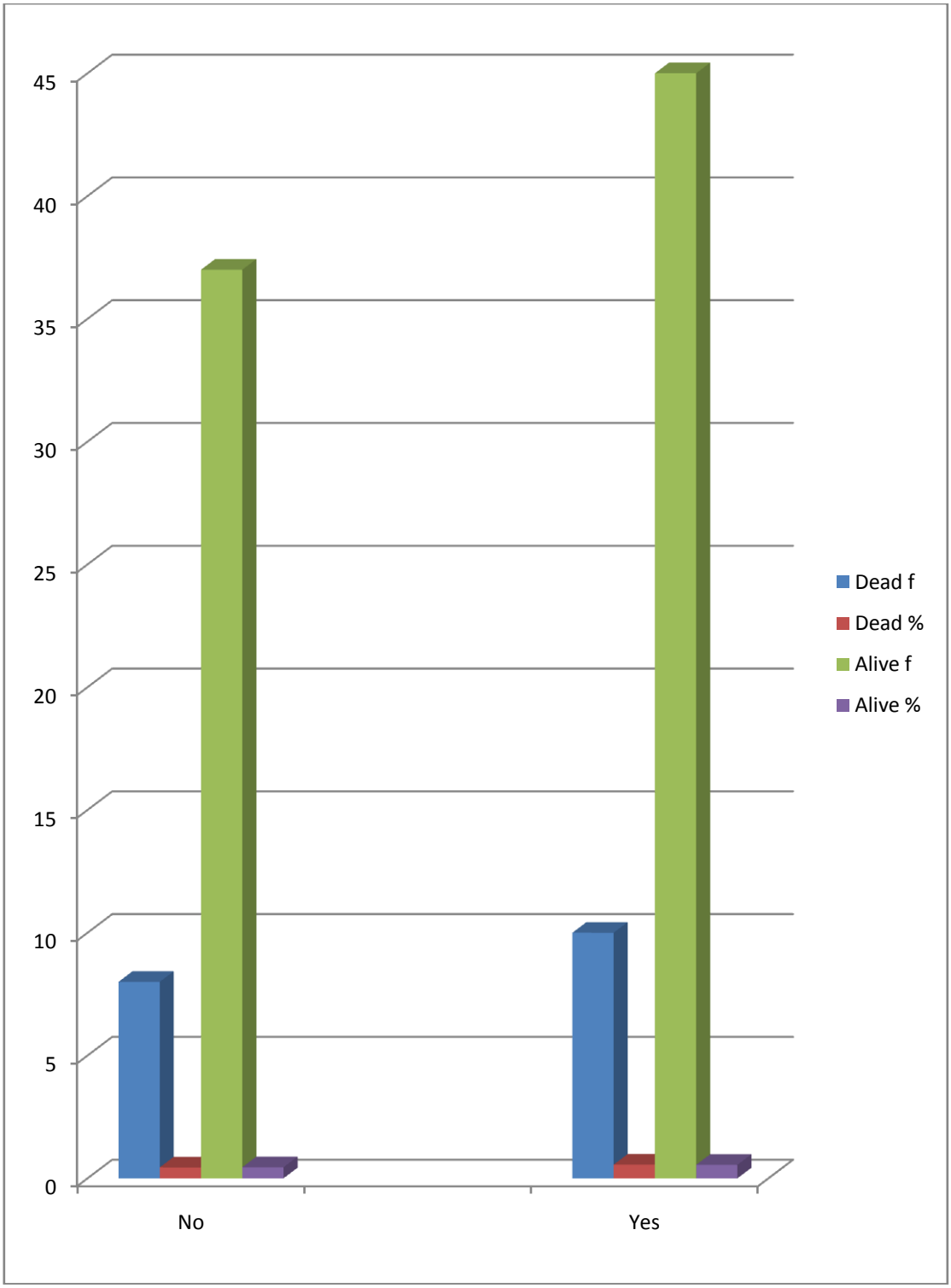
Alcohol	Dead		Alive		Total		Statistical inference
	f	*%*	*f*	*%*	*f*	*%*	
No	4	22.2%	28	34.1%	32	32.0%	X^2=0.964 Df=1 .326>0.05 Not Significant
Yes	14	77.8%	54	65.9%	68	68.0%	
Total	**18**	**100.0%**	**82**	**100.0%**	**100**	**100.0%**	



By using chi-square test overall outcome was compared with alcohol intake. In this study alcohol intake did not had significant effect on mortality.

TABLE – 16 CHI-SQUARE TEST TO COMPARE OUTCOME WITH SMOKING

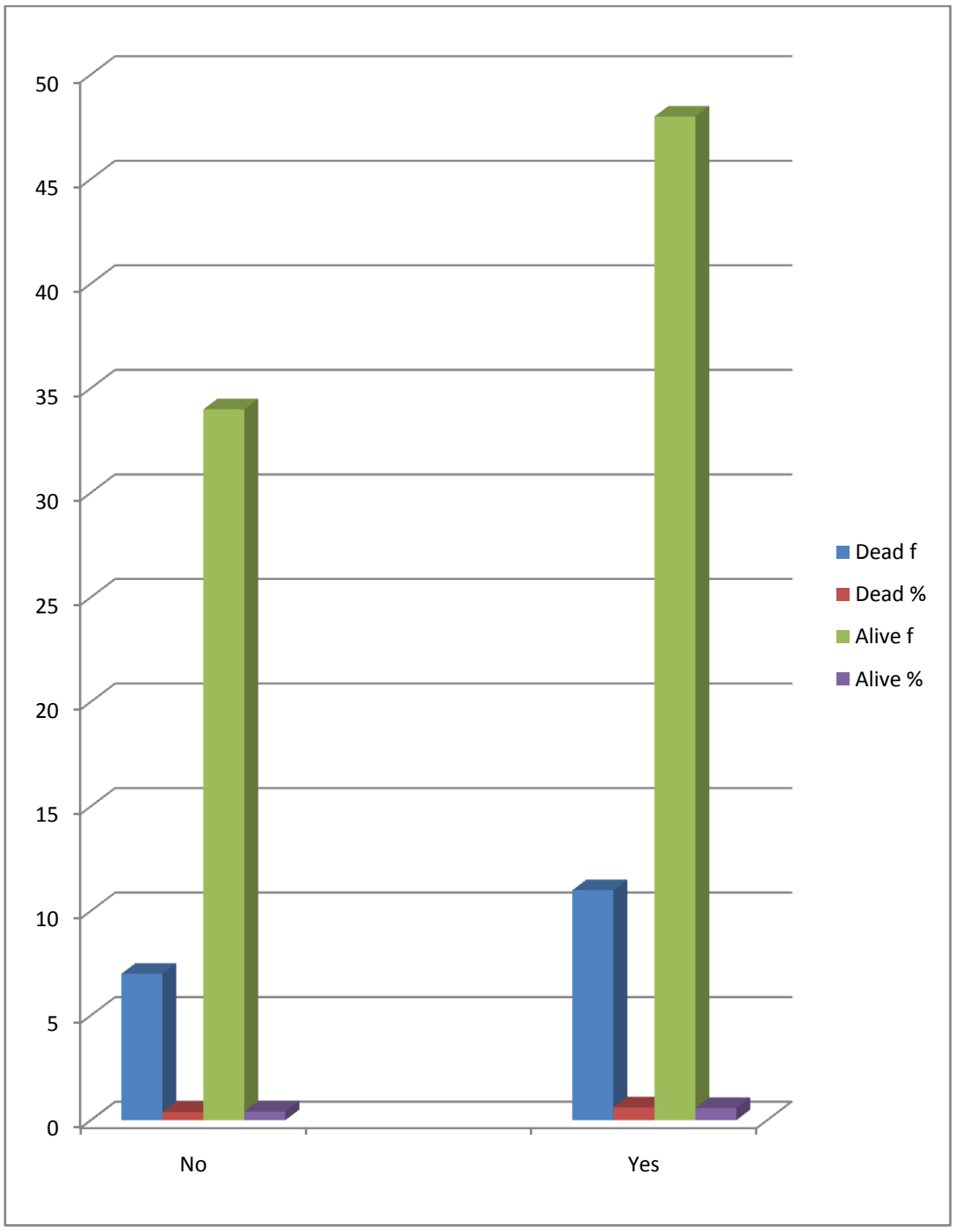
Smoking	Dead		Alive		Total		Statistical inference
	f	*%*	*f*	*%*	*f*	*%*	
No	8	44.4%	37	45.1%	45	45.0%	X^2=0.003 Df=1 .958>0.05 Not Significant
Yes	10	55.6%	45	54.9%	55	55.0%	
Total	**18**	**100.0%**	**82**	**100.0%**	**100**	**100.0%**	



Comparison of outcome with respect to smoking was done by using Chi-square test. It was found that smoking did not have significant effect on mortality in this study.

TABLE – 17 CHI-SQUARE TEST TO COMPARE OUTCOME WITH HYPERTENSION

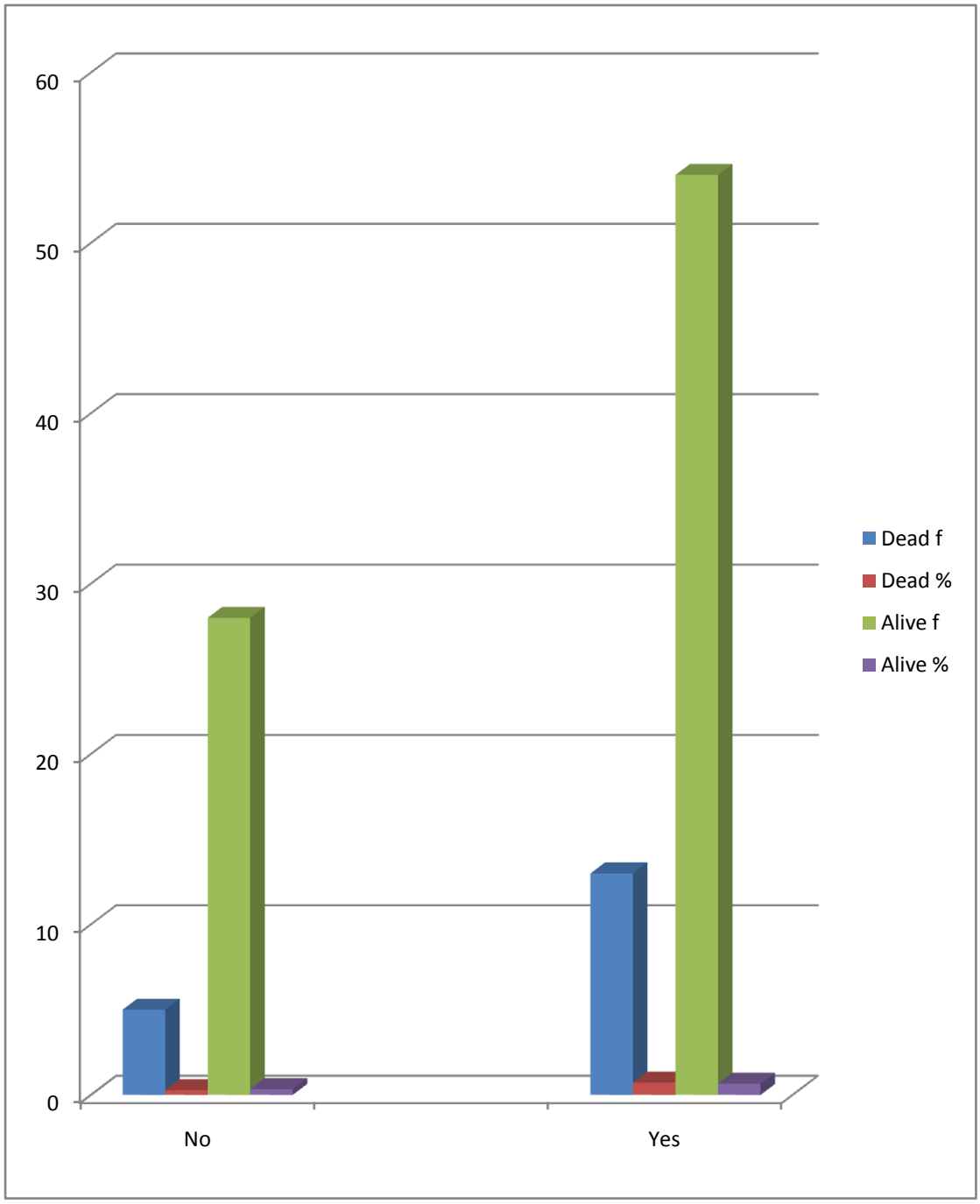
HT	Dead		Alive		Total		Statistical inference
	f	*%*	*f*	*%*	*f*	*%*	
No	7	38.9%	34	41.5%	41	41.0%	X^2=0.040 Df=1 .841>0.05 Not Significant
Yes	11	61.1%	48	58.5%	59	59.0%	
Total	**18**	**100.0%**	**82**	**100.0%**	**100**	**100.0%**	



Presence of Hypertension was compared with overall outcome using chi-square test. It was found that hypertension does not had significant effect on mortality in this study.

TABLE – 18 CHI-SQUARE TEST TO COMPARE OUTCOME WITH DIABETES MELLITUS

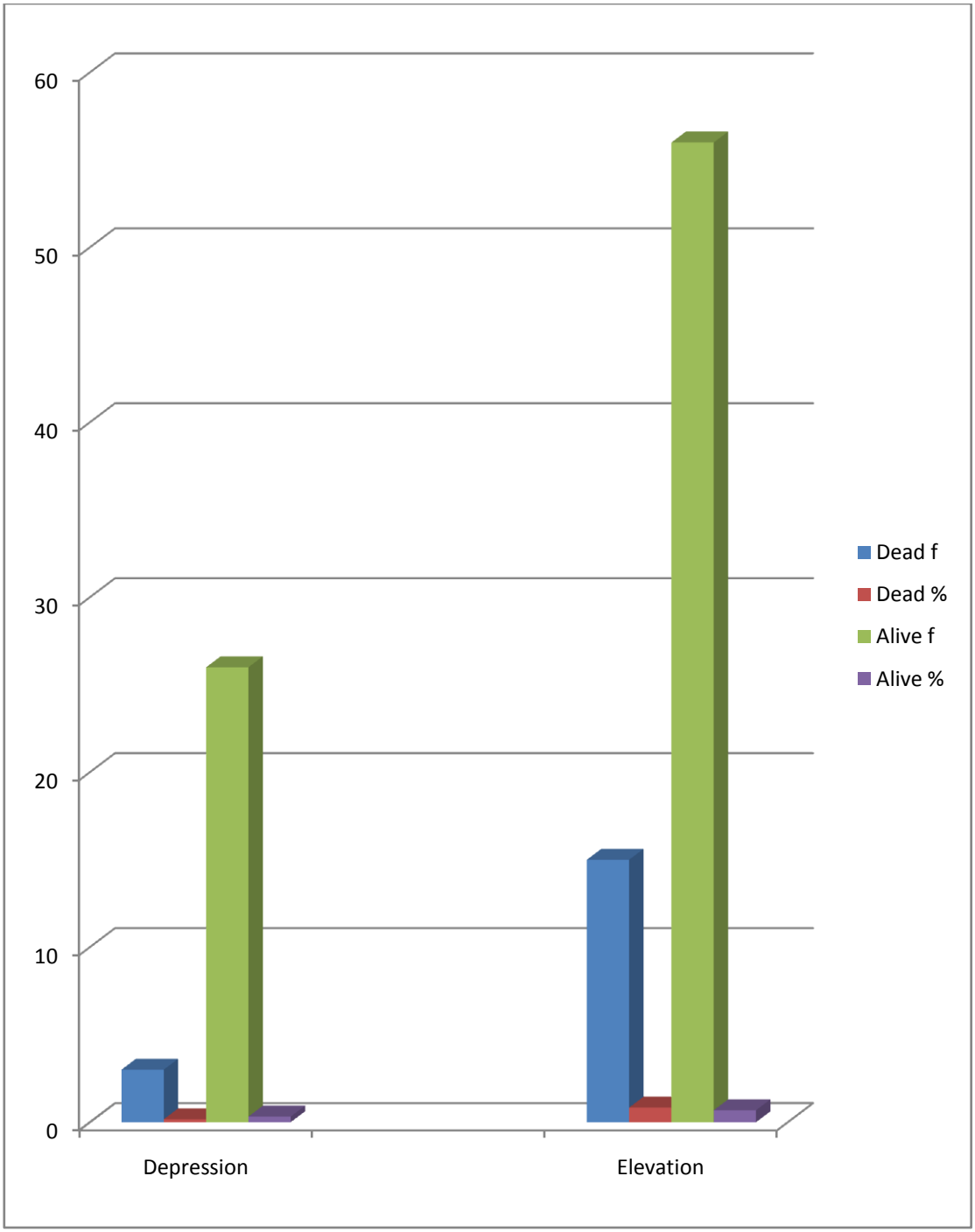
DM	Dead		Alive		Total		Statistical inference
	f	*%*	*f*	*%*	*f*	*%*	
No	5	27.8%	28	34.1%	33	33.0%	X^2=0.271 Df=1 .603>0.05 Not Significant
Yes	13	72.2%	54	65.9%	67	67.0%	
Total	**18**	**100.0%**	**82**	**100.0%**	**100**	**100.0%**	



By using chi-square test overall outcome was compared with the presence of Diabetes Mellitus. It was found that diabetes does not affect the outcome in this study.

TABLE 19 CHI-SQUARE TEST TO COMPARE OUTCOME WITH ST SEGMENT CHANGES AT PRESENTATION

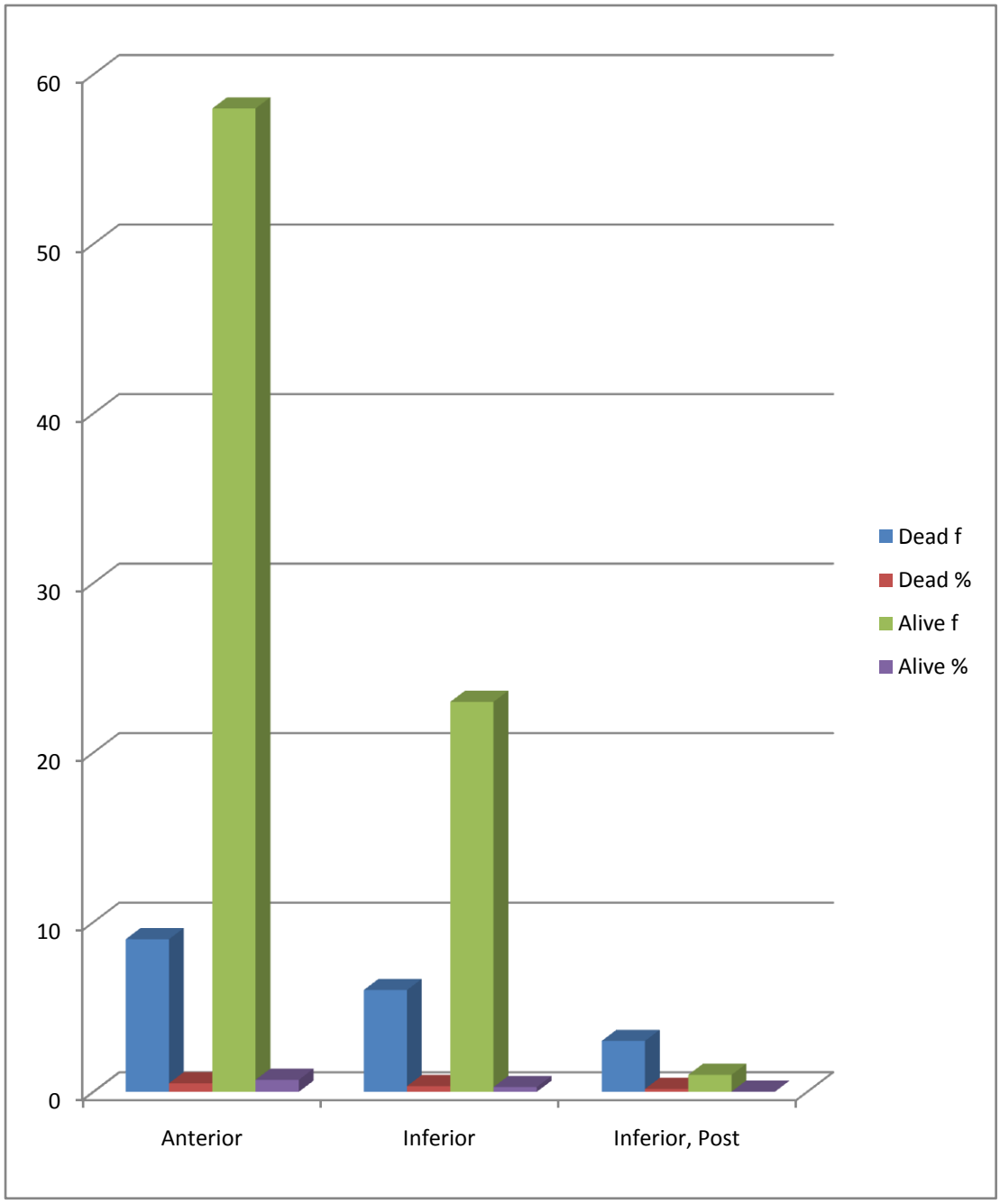
ST deviation	Dead		Alive		Total		Statistical inference
	f	*%*	*f*	*%*	*f*	*%*	
Depression	3	16.7%	26	31.7%	29	29.0%	X^2=1.622 Df=1 .203>0.05 Not Significant
Elevation	15	83.3%	56	68.3%	71	71.0%	



Chi – square test was used to compare the outcome with the ST segment changes at presentation. It was found that ST segment changes does not affect the outcome in this study.

TABLE – 20 CHI-SQUARE TEST TO COMPARE OUTCOME WITH THE WALL INVOLVED IN MI

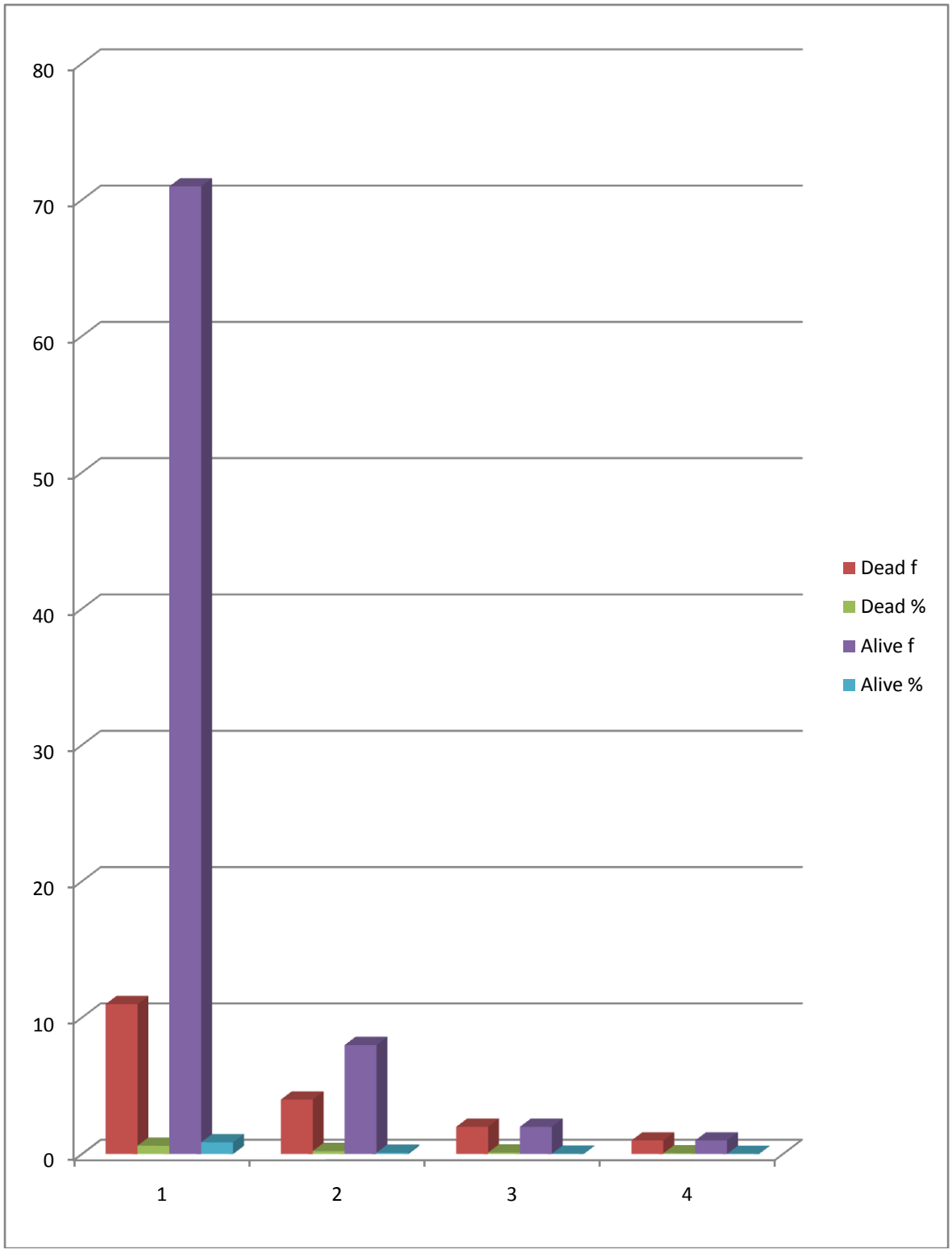
Wall involved	**Dead**		**Alive**		**Total**		**Statistical inference**
	f	***%***	***f***	***%***	***f***	***%***	
Anterior	9	50.0%	58	70.7%	67	67.0%	X^2=9.894 Df=2 .007<0.05 Significant
Inferior	6	33.3%	23	28.0%	29	29.0%	
Inferior, Post	3	16.7%	1	1.2%	4	4.0%	
Total	**18**	**100.0%**	**82**	**100.0%**	**100**	**100.0%**	



Using chi-square test overall outcome was compared with the wall involved in MI. It was found that the wall involved affect the prognosis significantly. Maximum death was observed in AWMI, followed by IWMI and the last was Combined Inferior+posterior wall MI.

TABLE – 21 CHI-SQUARE TEST TO COMPARE OUTCOME WITH THE TIME OF ONSET OF ARRHYTHMIA

Timing	Dead		Alive		Total		Statistical inference
	f	*%*	*f*	*%*	*f*	*%*	
1	11	61.1%	71	86.6%	82	82.0%	X^2=7.242 Df=3 .065>0.05 Not Significant
2	4	22.2%	8	9.8%	12	12.0%	
3	2	11.1%	2	2.4%	4	4.0%	
4	1	5.6%	1	1.2%	2	2.0%	
Total	**18**	**100.0%**	**82**	**100.0%**	**100**	**100.0%**	



Chi-square test was used to compare overall outcome with the time interval between the presentation and onset of arrhythmias. It was found that this timing does not affect the outcome significantly in this study.

TABLE – 22 CHI-SQUARE TEST TO COMPARE THE OUTCOME WITH THE TYPE OF ARRHYTHMIA

Type of arrhythmia	Dead		Alive		Total		Statistical inference
	f	*%*	*f*	*%*	*f*	*%*	
I DEGREE AVB	0	.0%	12	14.6%	12	12.0%	X^2=64.834 Df=11 .000<0.05 Significant
II DEGREE AVB	3	16.7%	6	7.3%	9	9.0%	
AF	0	.0%	4	4.9%	4	4.0%	
AIVR	0	.0%	13	15.9%	13	13.0%	
APC	0	.0%	5	6.1%	5	5.0%	
CHB	5	27.8%	1	1.2%	6	6.0%	
LBBB	1	5.6%	6	7.3%	7	7.0%	
RBBB	0	.0%	5	6.1%	5	5.0%	
SVT	0	.0%	1	1.2%	1	1.0%	
VF	3	16.7%	0	.0%	3	3.0%	
VPC	0	.0%	27	32.9%	27	27.0%	
VT	6	33.3%	2	2.4%	8	8.0%	
Total	**18**	**100.0%**	**82**	**100.0%**	**100**	**100.0%**	

Using chi-square test overall outcome was compared with various types of arrhythmias observed in this study. It was found that the type of arrhythmia significantly affect the mortality. Maximum death was observed with ventricular tachycardia (33.3%). Mortality percentage associated with various arrhythmias are CHB - 27.8% , , ventricular fibrillation – 16.7%, II degree AVB – 16.7%, LBBB – 5.6%.

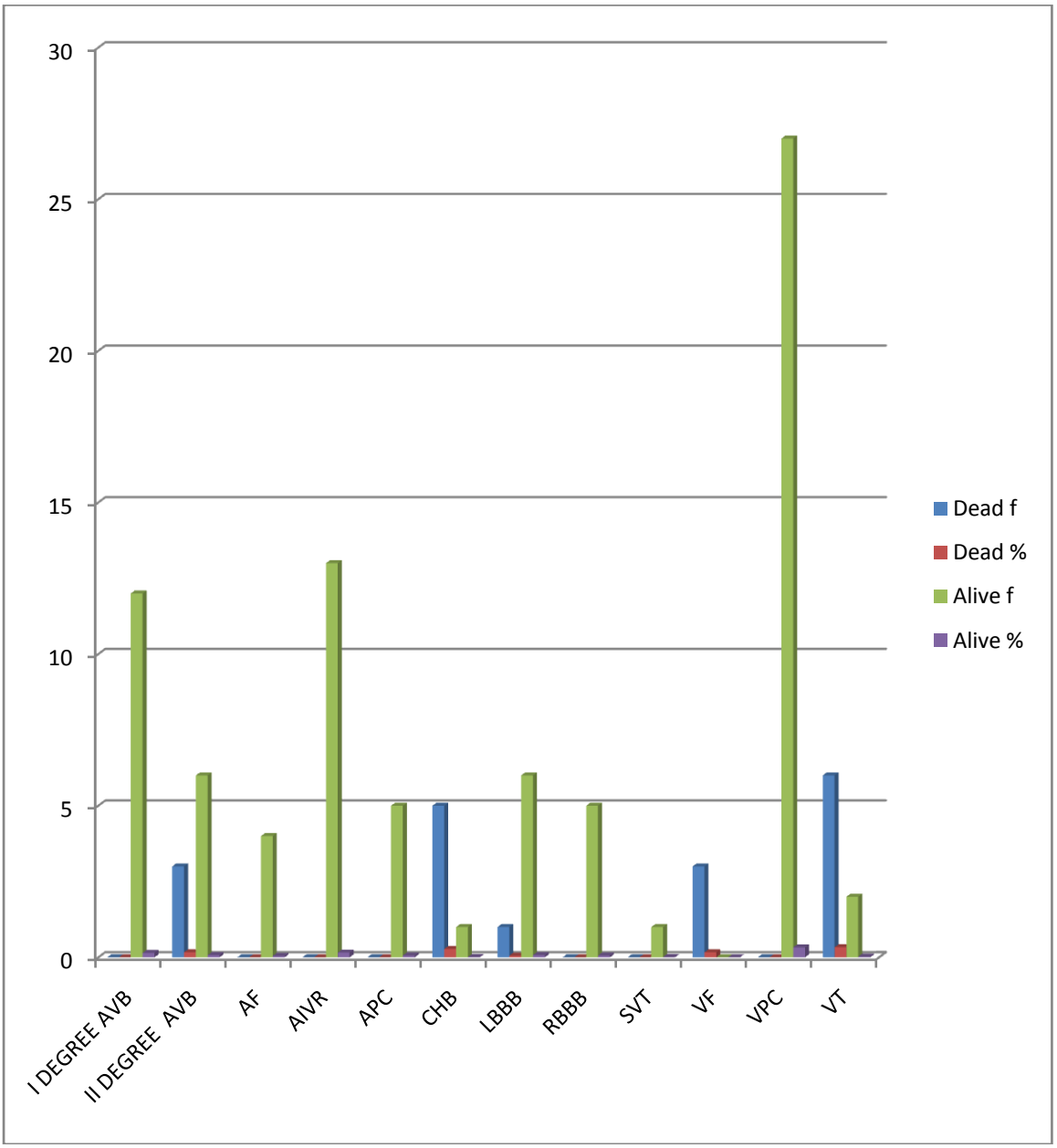
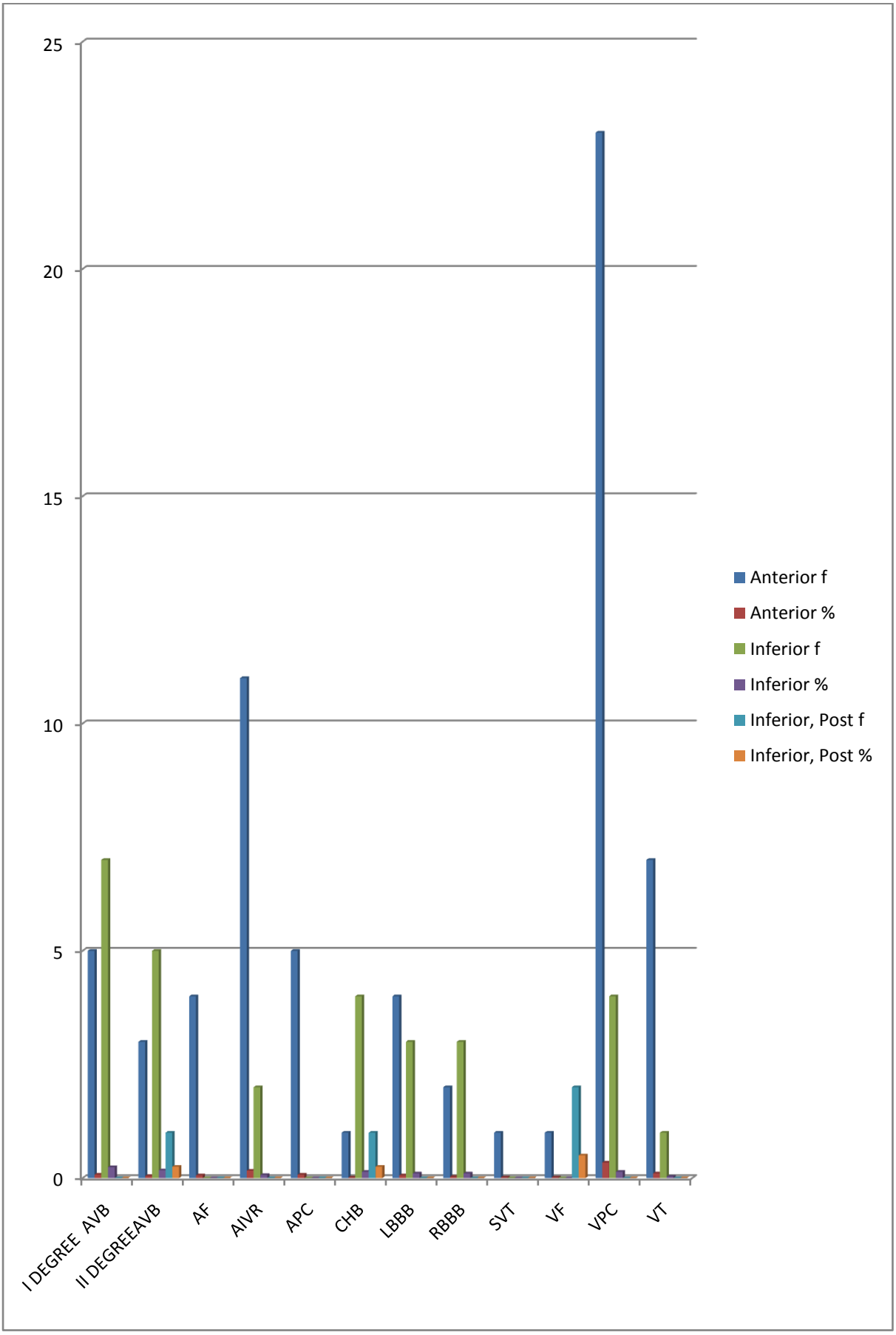


TABLE – 23 CHI-SQUARE TEST TO COMPARE THE TYPE OF ARRHYTHMIA WITH THE WALL INVOLVED

Type of arrhythmia	Anterior		Inferior		Inferior, Post		Total		Statistical inference
	f	*%*	*F*	*%*	*f*	*%*	*f*	*%*	
I DEGREE AVB	5	7.5%	7	24.1%	0	.0%	12	12.0%	X^2=64.464 Df=22 .000<0.05 Significant
II DEGREE AVB	3	4.5%	5	17.2%	1	25.0%	9	9.0%	
AF	4	6.0%	0	.0%	0	.0%	4	4.0%	
AIVR	11	16.4%	2	6.9%	0	.0%	13	13.0%	
APC	5	7.5%	0	.0%	0	.0%	5	5.0%	
CHB	1	1.5%	4	13.8%	1	25.0%	6	6.0%	
LBBB	4	6.0%	3	10.3%	0	.0%	7	7.0%	
RBBB	2	3.0%	3	10.3%	0	.0%	5	5.0%	
SVT	1	1.5%	0	.0%	0	.0%	1	1.0%	
VF	1	1.5%	0	.0%	2	50.0%	3	3.0%	
VPC	23	34.3%	4	13.8%	0	.0%	27	27.0%	
VT	7	10.4%	1	3.4%	0	.0%	8	8.0%	
Total	**67**	**100.0%**	**29**	**100.0%**	**4**	**100.0%**	**100**	**100.0%**	


25
20
15
10
5
0
I DEGREE AVB
II DEGREEAVB
AF
AIVR
APC
CHB
LBBB
RBBB
SVT
VF
VPC
VT
Anterior f
Anterior %
Inferior f
Inferior %
Inferior, Post f
Inferior, Post %

Using Chi-square test various arrhythmias were assessed for relation with the wall involved in MI. The pattern of arrhythmias seen with AWMI is as follows:

- VPC – 34.3%
- AIVR – 16.4%
- VT – 10.4%
- I degree AVB – 7.5%
- APC – 7.5%
- LBBB – 6%
- AF – 6%
- II degree AVB – 4.5%
- RBBB – 3%
- SVT – 1.5%
- VF – 1.5%
- CHB – 1.5%

The pattern of arrhythmias observed with IWMI is as follows:

- I degree AVB – 24.1%
- II degree AVB – 17.2%
- CHB – 13.8%
- VPC – 13.8%
- LBBB – 10.3%

- RBBB- 10.3%
- AIVR – 6.9%
- VT – 3.4%
- AF – 0%
- APC – 0 %
- SVT – 0%
- VF – 0 %

The pattern of arrhythmias observed with combined inferior and posterior wall MI is as follows:

- VF – 50%
- CHB – 25%
- II degree AVB – 25%
- VPC – 0 %
- APC – 0 %
- VT – 0%
- AIVR – 0 %
- I degree AVB – 0 %
- RBBB – 0 %
- LBBB – 0 %
- SVT – 0%
- AF – 0 %

DISCUSSION

In this study the total number of cases studied were 100. These cases were divided into five age groups. Distribution of age in them was as follows, 4 % of cases were in below 30 yrs age group. 15 % were in 31 – 39 yrs age group. A majority of 33 % belong to 40 – 49 yrs age group. 31 % were in 50 – 59 yrs age group.17 % were in above 60 yrs age group. (Table – 1)

In this study, 67 % of cases were male. 33 % cases were female. Male sex was predominant in the study group. (Table – 2)

Incidence of alcohol intake in this study population was 68 %. 32 % were non alcoholic. So the study population predominantly had alcoholics. (Table – 3)

Smoking frequency among the study population was 55 %. 45% of cases were non smokers. Hence this study included predominantly smokers. (Table – 4)

In this study group, prevalence of hypertension was 59 %. 41 % of the cases were non hypertensives. This shows predominance of hypertension among the affected people. (Table – 5)

In this study, prevalence of diabetes was 67 % . 33 % were non diabetic. Predominantly diabetics were the affected population. (Table – 6)

Comparing the difference in the presentation of ST changes, 71 % presented with ST elevation, 29 % presented with ST depression MI. (Table – 7)

Out of the 100 cases of acute MI studied, 67% had Anterior wall MI, 29 % had inferior wall MI. 4 % had combined involvement of both inferior and posterior wall. Hence anterior wall MI was the commonest, followed by inferior wall MI, and the least was combination of inferior and posterior wall MI. (Table – 8)

Usually majority of arrhythmias complicating acute MI occurs in the first 24 hours . T his was shown in a study by David T G et al in 1988[73] . Time interval between presentation and onset arrhythmias was studied in the study population. It was found that majority of arrhythmias occurred in the first day post MI which was 82%. 12 % cases developed arrhythmia in the second day. 4 % developed in the third day. Only 2% of cases developed arrhythmia on day 4. (Table – 9)

12 different types of arrhythmias were observed in the study population. Each occurred at a different frequency. VPC (27%)was clearly the most common arrhythmia observed. Followed by AIVR-13%, I degree AVB-12%, II degree AVB-9%,VT – 8%, LBBB- 7%, CHB – 6%, RBBB, APC each 5%,AF – 4%,VF – 3%, SVT- 1% respectively. Similar statistics was obsereved in a study by Goodman MJ , Lassers, Julian DG in 1984[74]. (Table – 10)

Out of the 100 cases of acute MI studied , 82 % of cases were alive, 18 % of cases died. (Table – 11)

Various biochemical parameters like age, BMI, Killip class, time interval between the presentation and the onset of arrhythmia, blood glucose, serum cholesterol, CPK – MB, urea, creatinine, sodium, potassium were analysed. (Table – 12)

Mean age was 49.97, standard deviation was 10.318. Mean BMI was 35.2390, SD was 4.58039. Mean Killip class was II, SD was 10123. Mean time interval between presentation and onset of arrhythmia was 1.26 days, with a SD of 0.630. Mean B.glucose was 183.46 with a SD of 59.214.

Mean S.cholesterol was 197.49 with a SD of 57.276. Mean CPK-MB was 51.02 with a SD of 19.944. Mean urea was 34.58 with a SD of 10.989. Mean creatinine was 0.8120 with a SD of 0.22351. Mean sodium was 139.27 with a SD of 2.981. Mean potassium was 4.2260 with a SD of 0.49536

Chi square test was used to assess the significance of age, sex, alcohol intake, smoking, diabetes mellitus, hypertension, ST changes, wall involved in MI, time interval between the presentation and onset of arrhythmias, type of arhhythmias in predicting the outcome, and also type of arrhythmias was compared with the wall involved.

Using chi-square test overall outcome is compared with the different age groups of the cases. It showed significant relationship between the age group and the outcome. Death was highest in 50- 59 years age group, and least in below 30 years age group. This is similar to the results obtained in a study by Recheard C. Klein, Zakauddin Vera, T, Mason, Davis, Calif, et al in 1984[75]. (Table – 13)

Chi – square test was used to compare overall outcome in different sex groups. It was found that sex had no significant effect on mortality in this study. (Table – 14)

By using chi-square test overall outcome was compared with alcohol intake. In this study alcohol intake did not had significant effect on mortality. (Table – 15)

Comparison of outcome with respect to smoking was done by using Chi-square test. It was found that smoking did not have significant effect on mortality in this study. (Table – 16)

Presence of Hypertension was compared with overall outcome using chi-square test. It was found that hypertension does not had significant effect on mortality in this study. (Table – 17)

By using chi-square test overall outcome was compared with the presence of Diabetes Mellitus. It was found that diabetes does not affect the outcome in this study. (Table – 18)

Chi – square test was used to compare the outcome with the ST segment changes at presentation. It was found that ST segment changes does not affect the outcome in this study. (Table – 19)

Using chi-square test overall outcome was compared with the wall involved in MI. It was found that the wall involved affect the prognosis significantly. Maximum death was observed in AWMI, followed by IWMI and the last was Combined Inferior+posterior wall MI. (Table – 20)

Chi-square test was used to compare overall outcome with the time interval between the presentation and onset of arrhythmias. It was found that this timing does not affect the outcome significantly in this study. (Table – 21)

Using chi-square test overall outcome was compared with various types of arrhythmias observed in this study. It was found that the type of arrhythmia significantly affect the mortality. Maximum death was observed with ventricular tachycardia (33.3%). Mortality percentage associated with various arrhythmias are CHB - 27.8% , , ventricular fibrillation – 16.7%, II degree AVB – 16.7%, LBBB – 5.6%. (Table – 22)

Using Chi-square test various arrhythmias were assessed for relation with the wall involved in MI. The pattern of arrhythmias seen with AWMI is as follows: VPC – 34.3%, AIVR – 16.4% , VT – 10.4%, I degree AVB – 7.5% , APC – 7.5%, LBBB – 6% , AF – 6% , II degree AVB – 4.5% , RBBB – 3% , SVT – 1.5% , VF – 1.5% , CHB – 1.5%.

The pattern of arrhythmias observed with IWMI is as follows:I degree AVB – 24.1% , II degree AVB – 17.2% , CHB – 13.8% , VPC – 13.8% , LBBB – 10.3% , RBBB- 10.3% , AIVR – 6.9% , VT – 3.4% , AF – 0% , APC – 0 % , SVT – 0% , VF – 0 %.

The pattern of arrhythmias observed with combined inferior and posterior wall MI is as follows: VF – 50% , CHB – 25% , II degree AVB – 25% , VPC – 0 % , APC – 0 % , VT – 0% , AIVR – 0 % , I degree AVB – 0 % , RBBB – 0 % , LBBB – 0 % , SVT – 0% , AF – 0 %. (Table – 23)

CONCLUSIONS

1. Myocardial infarction was most common in 40 – 49 years age group. Incidence is least in below 30 years age group. Majority of deaths was seen in 50 – 59 years age group.
2. Males were the predominant population and sex did not affect the prognosis significantly.
3. Alcoholics were predominant in the study group. Alcoholism does not affect the outcome.
4. Smoking was found in majority of the people. But it did not affect the outcome.
5. Diabetics were more compared to non- diabetics in the study population. It did not affect the outcome significantly.
6. Prevalence of hypertension was more in the study group. But it did not affect the outcome.
7. ST elevation MI was the predominant type of MI. but it does not affect the outcome significantly.
8. Anterior wall was the commonest to get involved in MI. Wall involved in MI affected the prognosis significantly. Maximum deaths occurred in AWMI.
9. Majority of arrhythmias occurred on day 1 post MI. the time interval between the presentation and onset of arrhythmias does not affect the prognosis significantly.

10. Among the various type of arrhythmias studied VPC was the most common. SVT was the least common. Mortality was affected significantly by the type of arrhythmia. Maximum death was observed in VT.
11. Overall mortality was 18 %.

LIMITATIONS

1. Period of study is minimal, due to time restrictions huge data could not be collected.
2. Study group is just adequate.
3. Biochemical parameters could not be assessed , due to lack of serial testing.
4. Late onset arrhythmias could not be studied since the study was conducted in ICCU and the duration of stay was limited.

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DATA COLLECTION PROFORMA

Name:

Age:

Sex:

Chief complaints:

Duration:

Risk factors :Smoking/Alcohol/Obesity/Dyslipidaemia/Gender

Comorbid conditions: HT/DM/Dyslipidaemia/CKD/COPD

Height :

Weight :

BMI:

BP:

PR:

General condition:

Type : STEMI/NSTEMI

Wall:

Thrombolysed/ Not thrombolysed:

Arrhythmias developed:

Investigations:

Treatment given:

Condition on discharge:

PATIENT INFORMATION LETTER

Name:

Age:

Sex:

Education:

Occupation:

Duration of illness:

Nature of illness:

Informant:

Relation with patient:

Education:

Occupation:

I, _____, is willing to include myself in the study titled “A Study on new onset arrhythmias in acute MI”

Signature

நோயாளி ஒப்புதல் படிவம்

நான் _____ மகாத்மா காந்தி அரசு நினைவு மருத்துவமனையில் மாரடைப்புக்கு சிகிச்சை பெற்று வருகிறேன். இந்த மருத்தவமனையில் நடைபெறும் ஆய்வான "மாரடைப்பினால் இதயத்துடிப்பில் ஏற்படும் கோளாறுகள்" பற்றி மருத்துவர் மூலம் எனது சொந்த மொழியில் அறிந்துகொண்டேன். இதில் பங்கேற்றுக்கொள்ள எனது சுயநினைவுடன் சம்மதம் அளிக்கிறேன்.

இடம் : கையொப்பம்

நாள் :

ETHICAL COMMITTEE CLEARANCE CERTIFICATE

K.A.P.VISWANATHAM GOVT. MEDICAL COLLEGE
TIRUCHIRAPALLI -1
INSTITUTIONAL ETHICS COMMITTEE

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Dr.N.Jothi,MD.,
Prof. and HOD of Anaesthesia,
K.A.P.V.Govt.Medical College, Trichy

LAW PERSON
Mr.R.Raveendran, ML
Rtd. District Judge

Mrs.Kalavathy,
Exnora Social Worker, Trichy

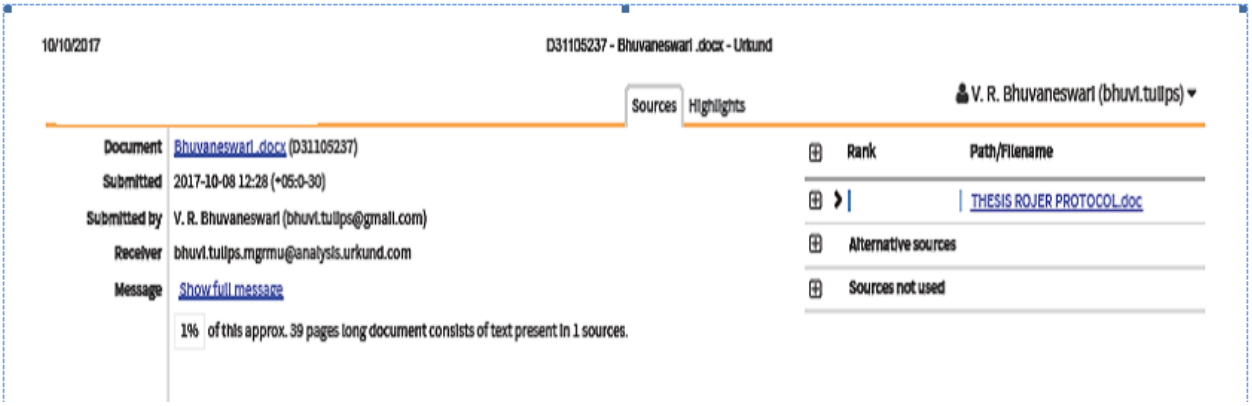
Smt.S.Gayathri,
Lay person.

This is to certify that the project work titled A Study on new onset arrhythmias in acute MI proposed by Dr.V.R.Bhuvaneswari, part of fulfillment of M.D/M.S course in the subject of General Medicine for the year 2015-2018 by The Tamilnadu Dr.MGR Medical University has been cleared by the Ethics committee.

29/12/2015

CHAIRMAN,
Institutional Ethics Committee
K.A.P.Viswanatham Govt. Medical College, Tiruchirapalli -1

PLAGIARISM REPORT



URKUND

Urkund Analysis Result

Analysed Document: Bhuvaneswari .docx (D31105237)
Submitted: 10/8/2017 8:58:00 AM
Submitted By: bhuvi.tulips@gmail.com
Significance: 1 %

Sources included in the report:

THESIS ROJER PROTOCOL.doc (D30282754)

Instances where selected sources appear:

1

ABBREVIATIONS

MI – Myocardial Infarction

STEMI – ST Elevation Myocardial Infarction

NSTEMI – Non ST Elevation Myocardial Infarction

AWMI – Anterior Wall Myocardial Infarction

IWMI – Inferior Wall Myocardial Infarction

PWMI – Posterior Wall Myocardial Infarction

HR – Heart Rate

BP – Blood Pressure

AVB – Atrio Ventricular Block

CHB – Complete Heart Block

VPC – Ventricular Premature contractions

APC- Atrial Premature Contractions

AF – Atrial Fibrillation

SVT – Supra Ventricular Tachycardia

VT – Ventricular Tachycardia

VF – Ventricular Fibrillation

AIVR – Accelerated Idio Ventrcular Rhythm

RBBB – Right Bundle Branch Block

LBBB – Left Bundle Branch Block

HT - Hypertension

DM – Diabetes Mellitus

BMI – Body Mass Index

SD – Standard Deviation

MASTER CHART

S.No	Age	Sex	Alcohol	Smoking	HT	DM	ST deviation	Wall involved	Timing	Type of arrhythmia	B.Glucose	s.cholesterol	CPK-MB	Urea	Creatinine	Sodium	Pottasium	Outcome
1	55	M	Y	Y	N	Y	ELEVATION	ANTERIOR	1	LBBB	241	123	56	34	0.8	142	3.7	1
2	52	M	Y	Y	Y	N	ELEVATION	INFERIOR	1	2 AVB	156	341	72	25	0.7	136	4.1	0
3	53	F	N	N	Y	Y	ELEVATION	ANTERIOR	1	AIVR	198	254	34	57	1.2	140	3.9	1
4	48	M	N	Y	N	Y	DEPRESSION	ANTERIOR	2	VPC	222	216	57	45	1.1	138	4	1
5	46	M	Y	Y	N	N	ELEVATION	INFERIOR	1	2 AVB	132	267	45	32	0.8	135	4.4	1
6	57	M	Y	Y	N	Y	ELEVATION	INFERIOR	1	CHB	302	167	68	37	0.7	145	5.1	0
7	59	M	Y	Y	Y	Y	ELEVATION	ANTERIOR	1	AIVR	178	178	42	54	1.4	135	4.3	1
8	54	M	Y	N	Y	Y	ELEVATION	ANTERIOR	1	SVT	216	113	26	45	1	137	3.6	1
9	49	M	Y	Y	N	N	ELEVATION	INFERIOR	1	RBBB	115	145	56	42	1.1	145	3.7	1
10	54	M	Y	Y	N	Y	DEPRESSION	ANTERIOR	4	VT	312	167	34	28	0.6	138	4.3	0
11	52	F	N	N	Y	Y	DEPRESSION	ANTERIOR	1	1 AVB	229	178	23	27	0.8	137	4.8	1
12	42	M	Y	N	N	Y	ELEVATION	INFERIOR	1	LBBB	278	190	57	34	0.9	145	3.8	1
13	39	M	Y	N	N	Y	ELEVATION	ANTERIOR	1	VPC	196	234	64	35	1	144	3.7	1
14	48	M	N	Y	Y	Y	ELEVATION	ANTERIOR	1	AF	105	265	34	27	0.6	134	4.7	1
15	29	M	Y	Y	N	N	ELEVATION	INFERIOR	2	1 AVB	114	276	38	18	0.6	135	4.9	1
16	59	M	Y	Y	Y	Y	ELEVATION	ANTERIOR	2	CHB	257	243	64	19	0.7	136	5	0
17	49	F	N	N	Y	Y	DEPRESSION	ANTERIOR	1	1 AVB	239	312	52	46	1	144	3.9	1
18	55	F	N	N	Y	Y	ELEVATION	INFERIOR	1	2 AVB	136	257	36	35	0.7	143	4.7	0
19	46	F	Y	N	Y	Y	ELEVATION	INFERIOR	1	2 AVB	194	213	45	48	1	134	4.6	1
20	38	M	Y	Y	N	Y	ELEVATION	ANTERIOR	1	AIVR	213	256	98	31	0.9	143	3.8	1
21	49	M	Y	Y	N	N	DEPRESSION	ANTERIOR	1	AF	301	210	23	23	0.8	136	4.6	1
22	58	M	Y	N	Y	N	ELEVATION	INFERIOR,POST.	3	VF	125	219	45	18	0.7	142	4.4	0
23	46	F	N	Y	Y	Y	DEPRESSION	ANTERIOR	1	APC	321	290	46	20	0.9	137	3.7	1

24	58	M	Y	N	Y	Y	DEPRESSION	ANTERIOR	1	VPC	246	221	76	22	0.8	138	4.8	1
25	47	M	Y	Y	Y	Y	ELEVATION	ANTERIOR	1	VPC	234	156	53	24	0.7	143	4.7	1
26	36	M	Y	Y	N	N	ELEVATION	INFERIOR	1	1 AVB	123	143	27	29	0.9	144	4.4	1
27	45	M	Y	N	N	Y	ELEVATION	ANTERIOR	2	VT	98	125	22	28	0.9	137	4.7	0
28	61	M	Y	N	Y	Y	ELEVATION	INFERIOR	1	2 AVB	249	198	35	38	1	142	3.8	1
29	55	M	Y	Y	Y	Y	ELEVATION	ANTERIOR	1	LBBB	156	167	59	57	1.2	143	4	1
30	58	F	N	N	Y	Y	ELEVATION	ANTERIOR	1	VF	278	178	64	24	0.7	136	4.6	0
31	47	F	N	N	N	Y	DEPRESSION	ANTERIOR	2	1 AVB	290	139	76	25	0.6	141	4.7	1
32	65	M	Y	Y	Y	N	ELEVATION	INFERIOR, POST.	1	CHB	152	198	56	31	0.7	138	5.2	0
33	48	M	Y	N	Y	Y	ELEVATION	ANTERIOR	3	VPC	193	195	45	36	0.9	137	3.8	1
34	53	F	N	N	Y	Y	ELEVATION	INFERIOR	1	1 AVB	218	145	52	47	1	142	4.4	1
35	62	F	N	N	N	Y	ELEVATION	ANTERIOR	1	AF	259	168	15	43	1	137	4.4	1
36	72	M	Y	Y	Y	Y	DEPRESSION	ANTERIOR	1	AIVR	290	178	97	27	0.7	141	4.8	1
37	65	F	N	N	Y	Y	DEPRESSION	ANTERIOR	1	VPC	241	147	57	59	1.4	137	4	1
38	46	M	Y	Y	N	Y	DEPRESSION	ANTERIOR	1	APC	246	190	53	52	1.3	137	3.7	1
39	51	M	N	Y	N	Y	ELEVATION	INFERIOR	1	RBBB	267	160	27	31	0.9	138	4.9	1
40	49	F	N	N	Y	N	ELEVATION	ANTERIOR	1	VPC	145	158	61	24	0.6	142	3.9	1
41	39	M	Y	Y	N	N	ELEVATION	ANTERIOR	1	VT	167	135	80	26	0.6	142	4.7	0
42	65	M	Y	Y	Y	Y	ELEVATION	ANTERIOR	1	APC	198	187	69	47	1.1	139	3.8	1
43	36	M	Y	Y	N	N	ELEVATION	INFERIOR	1	RBBB	167	168	51	38	0.9	135	4.8	1
44	55	F	N	N	Y	Y	ELEVATION	ANTERIOR	1	AIVR	191	147	42	32	0.8	136	4	1
45	46	F	N	N	Y	N	DEPRESSION	INFERIOR	1	LBBB	78	139	36	34	0.7	143	3.7	1
46	47	M	Y	Y	Y	Y	DEPRESSION	ANTERIOR	1	VT	109	156	43	32	0.7	142	4.9	1
47	33	M	Y	Y	N	N	ELEVATION	ANTERIOR	1	VPC	89	194	19	21	0.5	139	4.8	1
48	56	M	Y	Y	N	Y	ELEVATION	INFERIOR	2	CHB	153	134	50	32	0.7	136	5	0
49	56	M	Y	Y	N	Y	ELEVATION	ANTERIOR	1	VPC	183	193	57	21	0.5	141	3.6	1
50	49	M	Y	Y	Y	Y	DEPRESSION	ANTERIOR	1	AIVR	163	112	54	28	0.6	137	4.7	1
51	45	F	Y	N	Y	Y	ELEVATION	INFERIOR	1	1 AVB	111	135	46	47	1	142	4.9	1
52	46	M	N	Y	Y	Y	ELEVATION	ANTERIOR	2	VPC	189	135	75	21	0.6	142	4.4	1

53	38	M	Y	Y	N	Y	ELEVATION	INFERIOR	1	VT	156	149	34	24	0.5	138	3.9	0
54	62	M	Y	N	Y	Y	ELEVATION	ANTERIOR	1	LBBB	167	167	25	27	0.7	142	3.8	1
55	45	F	Y	N	Y	Y	ELEVATION	ANTERIOR	1	AIVR	189	178	67	30	0.7	139	4.8	1
56	56	F	N	N	Y	Y	ELEVATION	ANTERIOR	1	2 AVB	193	196	35	34	0.6	141	4.6	1
57	51	F	N	N	Y	Y	ELEVATION	INFERIOR	4	CHB	214	165	89	31	0.6	137	4.9	1
58	36	M	Y	Y	N	N	DEPRESSION	ANTERIOR	1	VPC	120	187	76	26	0.5	142	3.8	1
59	35	M	Y	Y	N	N	DEPRESSION	ANTERIOR	2	1 AVB	96	167	54	28	0.7	138	3.8	1
60	61	M	Y	N	Y	Y	ELEVATION	ANTERIOR	1	APC	214	123	23	25	0.6	137	4.7	1
61	26	M	Y	Y	N	N	ELEVATION	ANTERIOR	1	VPC	100	145	65	36	0.9	141	3.6	1
62	54	M	Y	Y	N	Y	ELEVATION	ANTERIOR	1	VT	321	178	45	32	0.8	138	4	0
63	43	F	N	N	Y	N	DEPRESSION	INFERIOR	1	1 AVB	214	290	34	31	0.8	136	4.8	1
64	45	M	Y	Y	N	Y	ELEVATION	ANTERIOR	1	VPC	243	136	89	45	0.9	135	4.4	1
65	49	M	Y	Y	Y	Y	ELEVATION	ANTERIOR	1	AIVR	256	301	70	47	0.9	141	3.4	1
66	52	M	Y	Y	N	Y	ELEVATION	ANTERIOR	1	APC	278	245	65	43	0.8	142	4.9	1
67	30	M	Y	Y	Y	N	ELEVATION	INFERIOR	1	LBBB	134	265	45	43	0.7	137	3.4	1
68	53	F	Y	N	Y	Y	DEPRESSION	ANTERIOR	1	AF	114	345	34	21	0.6	143	4	1
69	38	M	Y	Y	N	N	DEPRESSION	ANTERIOR	2	VPC	115	145	56	22	0.6	138	3.7	1
70	65	M	Y	N	Y	Y	ELEVATION	INFERIOR	1	VPC	178	135	78	32	0.8	135	3.8	1
71	39	M	Y	N	N	Y	ELEVATION	ANTERIOR	1	VPC	176	219	76	65	1.4	141	4.6	1
72	46	F	Y	N	N	Y	ELEVATION	INFERIOR	3	1 AVB	158	198	21	45	1	143	4.2	1
73	63	F	N	N	Y	Y	DEPRESSION	ANTERIOR	1	2 AVB	197	239	23	43	0.9	137	3.8	0
74	71	M	Y	N	Y	Y	DEPRESSION	ANTERIOR	1	VT	114	185	25	57	1.3	136	4.6	0
75	57	F	N	N	Y	Y	DEPRESSION	ANTERIOR	1	VPC	198	245	26	24	0.5	139	3.5	1
76	65	M	Y	N	Y	Y	ELEVATION	INFERIOR	1	AIVR	176	238	75	35	0.8	142	3.4	1
77	59	M	Y	N	Y	Y	ELEVATION	ANTERIOR	1	LBBB	145	301	45	31	0.7	144	4.8	0
78	57	M	Y	N	Y	Y	ELEVATION	INFERIOR	1	VPC	176	256	36	37	0.7	137	4.3	1
79	43	M	Y	Y	Y	N	DEPRESSION	ANTERIOR	1	AIVR	114	278	27	32	0.6	136	3.8	1
80	68	M	Y	N	Y	Y	ELEVATION	INFERIOR,POST.	2	VF	178	156	65	45	1	139	4	0
81	48	F	N	N	Y	N	ELEVATION	ANTERIOR	1	1 AVB	134	256	54	53	1.2	142	3.9	1

82	56	F	N	N	N	Y	DEPRESSION	ANTERIOR	1	VPC	156	269	56	43	1	143	5	1
83	44	M	Y	Y	Y	Y	ELEVATION	INFERIOR	1	AIVR	167	234	27	32	0.7	137	3.9	1
84	54	F	N	N	Y	N	DEPRESSION	ANTERIOR	1	VT	143	156	68	36	0.7	139	4.7	1
85	37	M	Y	Y	N	N	DEPRESSION	ANTERIOR	2	VPC	123	189	96	12	0.5	141	4.6	1
86	46	M	Y	Y	N	N	ELEVATION	ANTERIOR	1	VPC	167	167	45	19	0.5	143	3.8	1
87	52	F	N	Y	Y	N	ELEVATION	INFERIOR	1	VPC	178	134	67	35	0.9	136	3.3	1
88	64	F	N	Y	Y	N	ELEVATION	INFERIOR	3	CHB	134	112	78	48	1	142	4.1	0
89	36	M	Y	Y	N	N	DEPRESSION	ANTERIOR	1	AIVR	126	158	54	23	0.4	140	4.3	1
90	45	F	N	N	Y	N	ELEVATION	ANTERIOR	1	RBBB	189	178	32	25	0.5	137	4.8	1
91	67	F	N	N	Y	Y	ELEVATION	INFERIOR,POST.	1	2 AVB	191	187	14	26	0.5	137	3.9	1
92	57	F	N	N	Y	Y	DEPRESSION	ANTERIOR	1	VPC	173	176	56	43	0.9	140	3.7	1
93	19	M	Y	Y	N	N	ELEVATION	ANTERIOR	1	2 AVB	113	134	76	44	0.9	136	4.2	1
94	48	M	Y	Y	N	N	ELEVATION	ANTERIOR	1	VPC	156	159	56	52	1	142	4.1	1
95	38	M	Y	Y	N	Y	ELEVATION	INFERIOR	2	1 AVB	154	201	46	28	0.6	142	3.7	1
96	65	F	Y	Y	Y	Y	DEPRESSION	ANTERIOR	1	AIVR	97	357	78	40	0.9	137	3.8	1
97	63	F	N	Y	Y	Y	ELEVATION	ANTERIOR	1	RBBB	198	245	76	43	1	135	4.7	1
98	35	M	Y	Y	N	N	ELEVATION	ANTERIOR	1	VPC	207	278	37	27	0.6	140	3.5	1
99	47	M	Y	Y	N	N	ELEVATION	INFERIOR	1	VPC	109	216	45	30	0.7	137	3.6	1
100	41	M	N	Y	Y	N	ELEVATION	ANTERIOR	1	VPC	234	296	26	52	1.1	139	3.9	1

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